Physiology of exercise in health and disease, with special reference to effort intolerance, training and thermoregulation in man

-by-

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The original papers fall into three categories. Studies which (i) I was entirely responsible for initiating, conducting the research and writing the final paper. During the investigation I often received technical and other assistance, the papers, therefore, either appear under my own name with acknowledgement for help given or jointly with those giving the assistance named as co-authors. A.J. Sargeant whose name appears on several of the later papers was my technician (and later research assistant) for ten years. The publications aumbers are 1, 8, 11, 13, 18-20, 22-26, 28, 41-45 and 47-50.

(ii) I shared the responsibility of initiating and conducting the investigations with named co-authors. The studies in which my name appears first, I was the senior investigator and responsible for writing the paper. Publications 2-7, 9, 12, 14, 16, 17, 21, 27, 29-40 and 46.

(iii) I was partly responsible with named co-authors for the experimental observations presented. Publications are 10 and 25.

C.T.M. DAVIES

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imitations to the prediction of maximum oxygen take from cardiac frequency measurements

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AVIES, C. T. M. *Limitations to the prediction of maximum oxygen e from cardiac frequency measurements.* J. Appl. Physiol. 24(5): -706. 1968.—The cardiac frequency (f_H) and O_2 con- ption ($\dot{V}O_2$) curves of 80 healthy male subjects aged 20–50 s in relation to the over-all limitations of predicting maxi- n oxygen intake ($\dot{V}O_{2\,max}$) have been examined, together the accuracy of the Åstrand-Rhyming, Margaria, and itz-Wyndham prediction nomograms. The major draw- to all extrapolation methods is the asymptotic nature of O_2 curve. This introduces an error of 1,200 ml at the 95% idence level and precludes accurate and reliable estimation $O_{2\,max}$. In comparison, the error due to the intersubject ability in $f_{H\,max}$ is small and insignificantly different from to be expected from random day-to-day variations in surement of f_H and $\dot{V}O_2$. The mean errors with 95% confi- e limits using the three nomograms are -624 ± 836, 9 ± 776, and -430 ± 708 ml, respectively. The under- reasons which might be responsible for the asymptotic e of the f_H curve and alternative formulas for predicting $_{max}$ are discussed. It is concluded that if an accuracy ter than $\pm 15\%$ is required, then there is no alternative but easure $\dot{V}O_{2\,max}$ directly.

$\dot{V}O_{2\,max}$ prediction nomograms

E MAXIMUM OXYGEN INTAKE ($\dot{V}O_{2\,max}$) of an indi- al has often been used by exercise physiologists as an ctive measure of his capacity to perform hard physi- vork. Unfortunately, direct determination of $\dot{V}O_{2\,max}$ lves a series of difficult and sometimes dangerous riments. In order to obtain a reliable estimate of $_{max}$, two or three measurements are required at levels ork beyond the point at which the maximum rate of ntake occurs. In elderly people, cardiorespiratory nts, and sedentary subjects it is not always possible tain the motivation required, and often the exertion ved may be dangerous to the health of the indi- al. At least one patient has died on the treadmill g maximal effort (9). Indeed, few people in modern ty enjoy being run to exhaustion and some may actually fear the pain which accompanies physiological stress.

It is for these reasons that many exercise physiologists have sought to develop safe and reliable tests which, although they encourage the wider application of the concept of $\dot{V}O_{2\,max}$, avoid the drawbacks of procedures involving maximal effort. The most frequently suggested test in the literature is the one based on measurement of cardiac frequency (f_H) and oxygen consumption ($\dot{V}O_2$) at one or more rates of work; f_H is then plotted against $\dot{V}O_2$, and $\dot{V}O_{2\,max}$ is estimated by extrapolation of a fitted straight line (usually within a nomogram) to a theoretical maximum cardiac frequency (6). Some workers (17, 18) have proposed modifications to this procedure, but the essential premises of all methods which rely on submaximal f_H and $\dot{V}O_2$ measurements are the same, namely, *1)* the linear relationship between the two variables are linear up to and including maximum levels of work, and *2)* all subjects under consideration must be able to reach similar maximum cardiac frequency.

To the author's knowledge only Maritz and co-workers (18) have examined these premises and, although their work has clarified a number of points, their findings are difficult to interpret in several respects: *1)* their subjects were Bantu of small stature whose ages were unknown; *2)* the studies were conducted at an altitude of approximately 6,000 ft; *3)* during submaximal work f_H rarely reached values of 130 beats/min, the recommended minimum of Astrand and Rhyming (6) whose nomograms they compared with their own method; and *4)* at maximal work the mean observed $f_{H\,max}$ was 179 beats/min. This is extremely low by European standards. Their conclusion that $\dot{V}O_{2\,max}$ can be estimated with an accuracy of $\pm 8\%$ must be open to question. Serious doubts are known to surround the use of cardiac frequency as a test criterion. It can undergo random day-to-day variation quite independently of $\dot{V}O_2$ and is affected by temperature and training. All these factors could give rise to serious error and impair the accuracy of extrapolation procedures in the individual case.

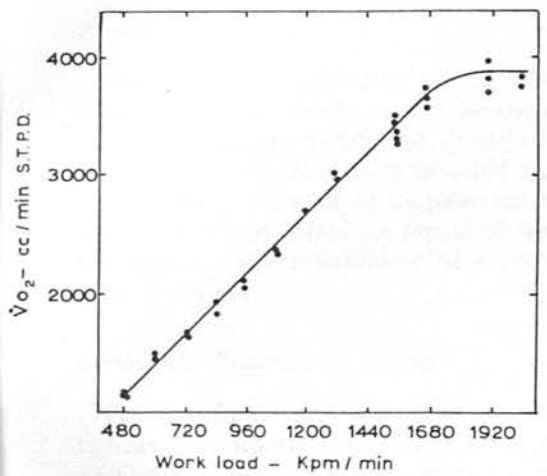
The present paper is concerned with the re-examination of the relationship between f_H and $\dot{V}O_2$ at submaxi-

ceived for publication 3 January 1968.

LE 1. *Anthropometric together with some ventilatory and circulatory data of 80 subjects grouped according to age*

Group	Age, yr	Weight, kg	Height, cm	$\dot{V}_E$ BTPS, liters/min	LA, mmoles/liter	$f_{H\,max}$, beats/min	$\dot{V}_{O_2\,max}$	
							liters/min	ml/kg
n = 22	22.3±1.8	69.1±8.8	176.4±6.8	131.9±16.5	14.1±3.4	195.6±5.9	3.5±0.4	50.7
n = 15	27.9±1.4	76.5±12.1	174.2±6.9	139.8±19.1	14.7±3.3	190.8±5.8	3.3±0.5	43.1
n = 12	33.4±1.6	78.2±9.2	176.5±5.8	138.8±19.2	13.2±2.1	188.0±4.8	3.6±0.6	46.0
n = 11	37.6±1.1	73.9±8.1	174.2±5.8	136.7±16.2	16.1±4.1	185.5±6.4	3.1±0.3	41.9
n = 10	42.9±1.5	81.8±10.6	179.0±7.0	133.1±24.3	13.8±1.3	184.1±4.4	3.1±0.5	37.8
n = 10	47.2±0.9	79.2±10.4	175.7±6.8	133.7±16.0	14.9±6.1	179.6±5.2	3.0±0.5	37.8

alues are means ± standard deviation.



. 1. Oxygen intake ($\dot{V}_{O_2}$) in relation to work load up to and ding maximum levels of work. Criterion for maximum oxygen e ($\dot{V}_{O_2\,max}$) in the present study was that three $\dot{V}_{O_2}$ values at lateau level should agree within ±5%. $\dot{V}_{O_2\,max}$ was taken as ıean of these three readings.

and maximal work loads on 80 male subjects aged 0 years, together with a reconsideration of the racy of the Astrand-Rhyming (6), Margaria (17), Maritz-Wyndham (18) prediction nomograms.

ERIALS AND EXPERIMENTAL PROCEDURE

ghty healthy male subjects whose physical details given in Table 1 were studied on the Müller bicycle meter. They reported to the laboratory following a lunch or breakfast and were asked to refrain from cing coffee and alcohol prior to the experiment.

ıring the preliminary period the subjects were al- d to become accustomed to the respiratory appa- and to pedaling the ergometer. The electrodes were in position and, if necessary, the subjects were ed to empty their bladders. A 6-min warmup at kpm/min was then given, followed by a 10-min ery period during which the subject rested quietly chair.

llowing this preliminary period the subjects exer- at 300, 600, 900, and either 1,050 or 1,200 kpm/ depending on the response of the subject to the penultimate work load, the idea being to span the f_H range of 110–170 beats/min for each subject. During the recovery period, which lasted for 10 min at the lighter work loads and 20 min at the higher levels of exercise, or until the cardiac frequency exceeded the resting rate by no more than 5 beats/min, whichever was the sooner, the subject rested in a chair placed at the side of the bicycle. No attempt was made to achieve absolute basal conditions either immediately prior to or following exercise. Throughout the exercise and recovery periods electric fans were directed at the subject and the temperature of the exercise room was kept approximately constant at 68 F.

Maximum oxygen intake was determined as a separate experiment after the method of Binkhorst and Van Leeuwen (8). The criterion for maximum values was that $\dot{V}_{O_2}$ should show no further rise with increasing work, i.e., at least three values at the plateau level must agree within ±5% (Fig. 1). In addition, blood lactate concentration during the 2nd min of recovery was taken as a guide to maximum effort but was never used as a criterion of maximal performance.

Two healthy male subjects were also studied at both submaximal and maximal effort on numerous occasions over a period of 6 months on the bicycle ergometer using similar procedures to those outlined above.

METHODS

Expired Air Volume

During submaximal work a low-resistance dry-gas meter (Parkinson Cowan Ltd.) was used in conjunction with rubber bellows to absorb peak flows (19) in a manner previously described (14). Inspiratory volume was continuously monitored via photocell relay switch on the meter and expired air was collected in Douglas bags. The meter was calibrated at frequent intervals by exercizing a subject into a steady state at several different work rates on the treadmill and then measuring the ventilatory volume with the meter and Douglas bag simultaneously. Corrections made for temperature and difference in N_2 content of inspired and expired gas following the emptying of the bag contents through a standard Tissot spirometer.

During maximal work the subjects breathed directly

through a McKerrow-Otis low-resistance valve into short lengths of smooth 1½-inch internal bore tubing and wide-diameter vinyl plastic Douglas bags. The very low resistance of the collection circuit (<1 cm H_2O) may account for the very high ventilatory volumes recorded in the subjects. It was not uncommon to find values in excess of 180 liters/min BTPS at the third supramaximal work load in both the young and the older subjects. Indeed, it will be noted that there was no tendency for either ventilatory volume ($\dot{V}_E$) or blood lactic acid (LA) concentration to decline with increasing age in this investigation (Table 1).

Expired Air Analysis

The Servomex paramagnetic O_2 and Beckman CO_2 infrared analyzers were used for the analysis of expired air samples. Gas was transferred directly into either small vinyl plastic sampling bags or oiled syringes. The analyzers were calibrated at frequent intervals against the standard (Lloyd) Haldane chemical method. Details of the accuracy and reliability of the method have already been given (14).

Cardiac and Respiratory Frequency

Cardiac frequency was recorded continuously using lightweight clip electrodes (12) in conjunction with an ECG differential amplifier and linear instantaneous rate meter (20). Respiratory frequency was monitored from a bead thermistor placed in the opening of the mouthpiece (13).

Blood Lactic Acid

Blood samples were taken from a prewarmed fingertip 2 min after the cessation of exercise and then analyzed for lactic acid using the Ström (23) modification of the Barker-Summerson method (7).

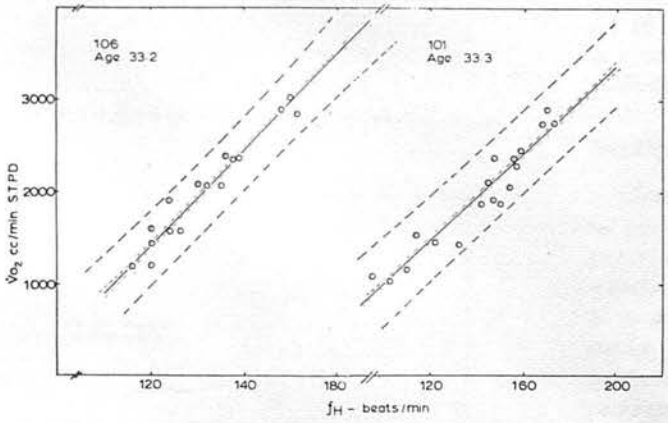


FIG. 2. Regression of cardiac frequency (f_H) on oxygen intake ($\dot{V}_{O_2}$)—95% confidence limits for the line and 95% confidence limits for a new observation.

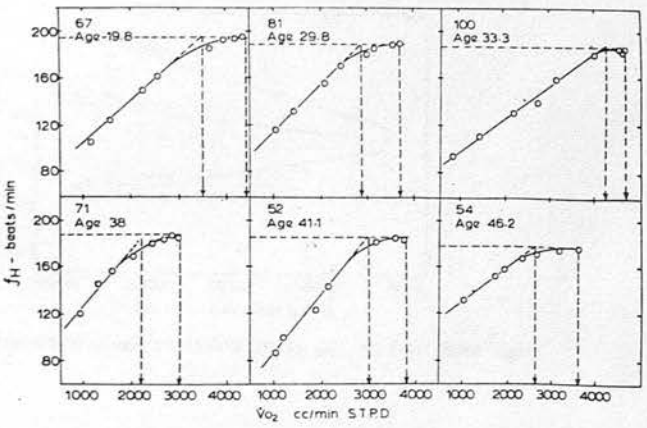


FIG. 3. Cardiac frequency (f_H) in relation to oxygen intake ($\dot{V}_{O_2}$) at various work rates up to and including maximum exercise for subjects of different age.

RESULTS

Relationship of Cardiac Frequency and Oxygen Consumption

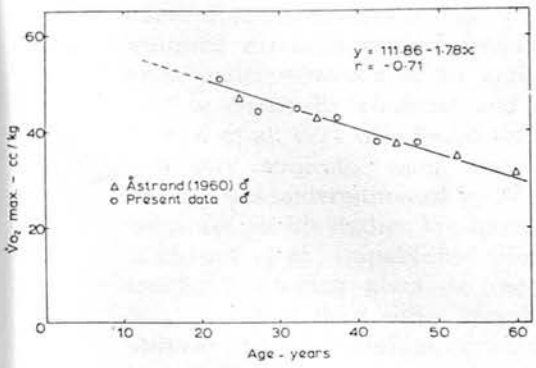
At submaximal work loads—random errors and physiological variability. To assess the contribution of both random errors and physiological variability in interfering with the accurate prediction of $\dot{V}_{O_2\,max}$, a large number of measurements of f_H and $\dot{V}_{O_2}$ were made on two subjects during work on the bicycle ergometer over a 6-month period (Fig. 2). The contribution of random error due either to natural day-to-day physiological variation or error of measurement can be assessed from the 95% confidence limits for any new observation. It is of the order of ±500 ml for the two subjects. The standard deviation about the regression line is ~190 ml and this will constitute an error of ~±7% in the determination of $\dot{V}_{O_2\,max}$, assuming that the $f_H\dot{V}_{O_2}$ is extrapolated to a $f_{H\,max}$ of 190 beats/min. This error alone (which, it is worth noting, was obtained on subjects well versed with experimental procedure and to working on the bicycle ergometer) is similar to the over-all error for the method reported by Maritz and co-workers and presents a limitation to the prediction of $\dot{V}_{O_2\,max}$ from simple measurements of cardiac frequency.

At maximal work loads. Figure 2 shows that a linear relationship between f_H and $\dot{V}_{O_2}$ does exist over most of its working range. However, at near maximum effort, as first shown by Wyndham et al. (26), the relationship breaks down and the curve becomes asymptotic (Fig. 3). This nonlinearity in the f_H curve occurs irrespective of age although with a high $\dot{V}_{O_2\,max}$ there was some reduction in the response (cf. Fig. 3 *subject 100*, and Fig. 7). Thus, in all groups of subjects investigated in this study, extrapolation of the $f_H/\dot{V}_{O_2}$ line to an observed $f_{H\,max}$, the $\dot{V}_{O_2}$ will always be less than the observed $\dot{V}_{O_2\,max}$ value at the asymptote. The magnitude of the underestimation can be seen from Fig. 3 and for the present data it is of the order of 1,200 ml at the 95% confidence level. This, compared to the error which is likely to

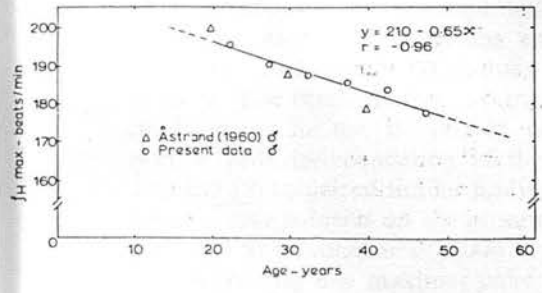
ccur from random variation (Fig. 2), is highly signifi-
nt ($P > 0.001$) and constitutes a serious limitation to
l methods which aim to predict $\dot{V}O_2$ from data col-
cted at submaximal levels of exercize.

Interindividual differences in $f_{H\ max}$. The regressions of $\dot{V}O_{2\ max}$ and $f_{H\ max}$ on age are shown in Figs. 4 and 5, spectively. They agree well with data already reported the literature (1–3). Cardiac frequency falls at a rate ~10 beats/decade and clearly must be taken into count when using extrapolation procedures. However, totally different picture arises if one studies the inter-lividual variation in $f_{H\ max}$ within each given age oup (Fig. 6).

In young adult subjects aged 20–25 years $f_{H\ max}$ shows small but significant negative correlation with $\dot{V}O_{2\ max}$ ig. 6), but with increasing group age the relationship idually changes so that in the oldest group the correla-n becomes decidedly positive. Thus, in young subjects th a high observed $\dot{V}O_{2\ max}$ the error of underestima-n from the asymptotic nature of the $f_H/\dot{V}O_2$ curve will d to be canceled out by the error produced by the fall $f_{H\ max}$. In young sedentary and older well-trained jects the reverse will be true; the errors from the two rces will combine to produce a larger underestimation $\dot{V}O_{2\ max}$. These errors could be important in the indi-ual case. However, it must be borne in mind that hin a single group the interindividual variation of ax about a population mean is not large. The coeffi-t of variation of $f_{H\ max}$ for all groups is ~5% and, of lf, the error that is likely to be produced by variation



G. 4. Maximum oxygen intake ($\dot{V}O_{2\ max}$) in relation to age.



5. Maximum cardiac frequency ($f_{H\ max}$) in relation to age.

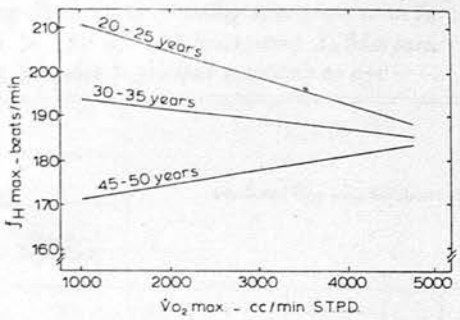


FIG. 6. Interindividual variation in $f_{H\ max}$ within three age groups.

in an individual's $f_{H\ max}$ is small. Extrapolating the $f_H/\dot{V}O_2$ line to an observed $f_{H\ max}$ instead of the population mean $f_{H\ max}$ for each group as given in Fig. 5 results in an error which is insignificantly different from that to be expected from random day-to-day variation in the measurement of f_H and $\dot{V}O_2$ and asymptotic nature of cardiac frequency curve.

Over-all errors in estimation of $\dot{V}O_{2\ max}$. The over-all errors in the estimation of $\dot{V}O_2$ by three standard methods are given in Tables 2 and 3. As is to be expected, all procedures produced a significant bias toward underestimation of $\dot{V}O_{2\ max}$. Using single measurements of f_H at relatively low work loads ($f_H > 120 < 150$ beats/min) this factor is combined with a large nonsystematic error. Using the Åstrand nomogram under these conditions, 95% probability limits are such that the predicted value of $\dot{V}O_{2\ max}$ will be within ±1.22 liters/min of the observed value. Thus, for a predicted value of 3.5 liters/min the true value may lie anywhere between 1.44 and 3.86 liters/min, clearly a very crude estimate of $\dot{V}O_{2\ max}$. Using, however, a higher value of f_H (>165 beats/min), or two f_H values (which cover the 130–170 beats/min range), or the calculated regression of $\dot{V}O_2$ on f_H, this estimation can be improved significantly. The mean errors and standard deviation of the estimate are − 624 ± 420, −529 ± 390, and −430 ± 356 ml, respectively. In the latter case this is equivalent to a percent error of 14 ± 11%. Or in terms of probability it would be expected for an observed $\dot{V}O_{2\ max}$ of 3.5 liters/min, 5% of individuals would lie outside the range 2.5–3.8 liters/min.

DISCUSSION

Clearly, the premises *1*) a linear relationship between f_H and $\dot{V}O_2$ and *2*) the ability of all subjects within a particular age group to reach similar $f_{H\ max}$ values on which the prediction of $\dot{V}O_{2\ max}$ from submaximal values of f_H and $\dot{V}O_2$ are based have not been validated by this study. However, the error arising from *2*) is small and insignificantly different from that to be expected from random day-to-day variation in the estimation of $\dot{V}O_{2\ max}$ from measurements of f_H and $\dot{V}O_2$. The major limitation to direct prediction of $\dot{V}O_{2\ max}$ from f_H and $\dot{V}O_2$ data would seem to be the asymptotic nature of the $f_H/\dot{V}O_2$

TABLE 2. *Relationship of observed to predicted $\dot{V}O_{2\ max}$ using 1) Åstrand-Rhyming nomogram with $f_H > 120 < 140$ beats/min; 2) Åstrand-Rhyming nomogram with $f_H > 140 < 180$ beats/min; 3) Margaria nomogram, and Maritz-Wyndham extrapolation procedure in 80 subjects grouped according to age*

Group	$\dot{V}O_{2\ max}$, liters/min STPD					Predicted $\dot{V}O_{2\ max}$ to Observed $\dot{V}O_{2\ max}$, liters/min STPD			
	Observed	Predicted							
		Åstrand-Rhyming $f_H > 120 < 140$	Åstrand-Rhyming $f_H > 140 < 180$	Margaria	Maritz-Wyndham				
	1	2	3	4	5	2 − 1	3 − 1	4 − 1	5 − 1
I	3.52±0.40	2.54±0.63	2.88±0.44	2.83±0.50	2.97±0.46	−0.96±0.47	−0.64±0.39	−0.69±0.41	−0.55±0.40
II	3.31±0.45	2.68±0.75	2.96±0.45	2.94±0.41	2.94±0.43	−0.63±0.66	−0.35±0.39	−0.37±0.35	−0.37±0.34
III	3.60±0.57	2.66±0.64	2.93±0.66	3.15±0.80	3.21±0.62	−0.94±0.48	−0.67±0.40	−0.45±0.43	−0.39±0.43
IV	3.14±0.29	2.28±0.61	2.44±0.29	2.62±0.45	2.68±0.33	−0.86±0.67	−0.70±0.30	−0.52±0.39	−0.46±0.21
V	3.09±0.49	2.31±0.25	2.31±0.33	2.65±0.26	2.68±0.36	−0.78±0.28	−0.78±0.31	−0.44±0.33	−0.41±0.29
VI	3.05±0.46	2.22±0.32	2.28±0.31	2.48±0.34	2.56±0.37	−0.83±0.58	−0.77±0.48	−0.57±0.43	−0.49±0.50

Value are means ± standard deviation.

TABLE 3. *Over-all relationship of predicted less observed $\dot{V}O_{2\ max}$ in the 80 subjects*

Subj	Predicted Less Observed $\dot{V}O_{2\ max}$, ml/min STPD			
	Åstrand-Rhyming nomogram		Margaria nomogram	Maritz-Wyndham
	$f_H > 120 < 140$, beats/min	$f_H > 140 < 180$, beats/min		
Males	−843±613	−624±420	−529±390	−430±356

Values are means ± standard deviation.

curve. Extrapolation of the $f_H/\dot{V}O_2$ line to an observed or assumed maximal cardiac frequency will result in a gross underestimation of an individual's $\dot{V}O_{2\ max}$. This finding essentially confirms and extends the work of Rowell et al. (21) who found that for groups of young sedentary subjects, using the Åstrand nomogram, $\dot{V}O_{2\ max}$ was underestimated by 27 ± 7%. Rowell used a treadmill for his studies. We have recently shown (R. J. Shephard et al., unpublished observations) that uphill treadmill running gives on the average 7% higher $\dot{V}O_{2\ max}$ values than either bicycling or step climbing, although the relationships between submaximal f_H and $\dot{V}O_2$ measurements remain essentially unchanged in all three forms of exercise. Thus, if the treadmill had been used in the present study, the error of underestimation (Table 2) might have been expected to increase to ~30% using the Åstrand nomograms and ~22% using the Margaria and Maritz-Wyndham extrapolation procedures. The result of this investigation shows that the nonlinearity in the $f_H/\dot{V}O_2$ at near maximal efforts occurs quite independently of the age of the subject. Åstrand (2) criticized similar findings of Wyndham et al. (26) on four subjects on the grounds that the data were collected at an altitude of 6,000 ft. An asymptotic heart rate curve and low maximal pulse have been shown by Åstrand and Åstrand (4) in subjects suffering from tissue hypoxia. However, since London is a lowland city, this criticism is unlikely to be applied to my results.

The uptake of oxygen is governed quantitatively by the equation

$$\dot{V}O_2\ (ml/min) = \text{cardiac frequency (beats/min)} \times \text{stroke volume (ml/beat)} \times (A\text{-}\dot{V}O_2 \text{ difference (vol \%)}$$

If, as has been shown (5), no further rise in stroke volume takes place beyond a $f_H \sim 120$ beats/min, then at higher rates of work a further increase in $\dot{V}O_2$ will depend solely on the cardiac frequency and arteriovenous difference. There is no direct evidence in man to indicate which of these two variables is the first to reach limiting values at maximum effort. The results of this study, however, would suggest that it is cardiac frequency and that a small amount of extra oxygen is extracted by the muscles from the circulatory blood by widening the $(A\text{-}\dot{V})O_2$ difference. Donald et al. (15) have shown this to occur in patients who suffer from mitral stenosis and find difficulty in increasing their cardiac output during exercise. It is possible that a similar compensatory mechanism occurs in health, especially in sedentary subjects due to a gradual shutdown of the splanchnic region at near maximum effort (24). The resulting redistribution of cardiac output from areas of low to those of high O_2 extraction may lead to a further unsaturation of venous blood and a rise in $\dot{V}O_2$ without a concomitant increase in heat output.

It must be emphasized that the present study was carried out on sedentary subjects who, prior to measurement, had minimum training on the bicycle ergometer. Probably one of the main effects of short-term training on the cardiovascular system is regulatory leading to an optimization of cardiac output distribution (16). At submaximal work loads the athlete exercizes with a lower cardiac output for a given $\dot{V}O_2$ compared with untrained subjects due to a more effective redistribution of blood from nonexercizing regions to the working muscles. At near maximum effort the volume of blood in areas of low O_2 extraction may well be small and the effect of a

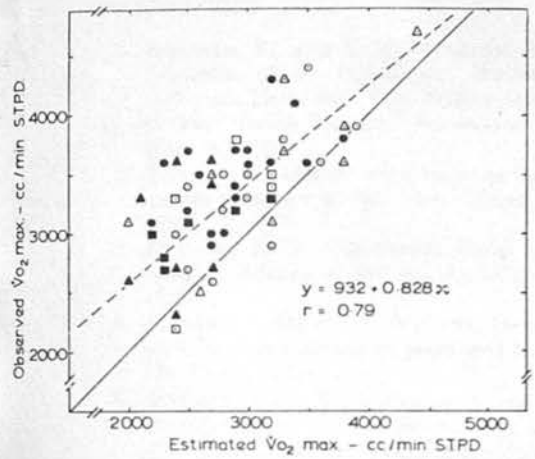


FIG. 7. Relationship of predicted to observed $\dot{V}O_{2\,max}$. Note that the higher the value of observed $\dot{V}O_{2\,max}$ the greater is the accuracy of prediction from f_H and $\dot{V}O_2$ measurements. Group I ●, group II ○, group II ●, group III △, group IV ■, group V □, group VI ▲.

shutdown on the $f_H/\dot{V}O_2$ curve may be marginal. We are in the process of completing investigations into this problem at the present time (Davies, Tuxworth, and Young, unpublished observations). Further arterial desaturation may take place in highly trained subjects. Rowell et al. (22) found that during a 3-min run to exhaustion, arterial oxygen saturation fell from 95.8% at rest to 93.4 and 91.4% in a sedentary group before and after training and to 85.2% in endurance athletes. This would tend to offset changes in the unsaturation of venous blood and, thus, may lead to narrowing rather than widening of the $(A\text{-}\dot{V})O_2$ difference. The same authors have also reported a 50% reduction in the underestimation of $\dot{V}O_{2\,max}$, using the Åstrand-Rhyming nomogram following a 6-month period of training.

Accuracy of $\dot{V}O_{2\,max}$ Prediction

The importance of the intraindividual variations in f_H and $\dot{V}O_2$ on the accuracy of $\dot{V}O_{2\,max}$ prediction has been stressed by Wyndham (25). At low work loads the coefficient of variation (cv) of f_H is high and when single measurements are used as in the Åstrand-Rhyming nomogram, the 95% confidence limits for any given predicted value of $\dot{V}O_{2\,max}$ are wide. However, contrary to Wyndham's finding, the cv of f_H around $\dot{V}O_2$ is not constant. At the lower work loads the intraindividual variation of f_H and $\dot{V}O_2$ is ~8 and ~3%, respectively (Fig. 1), but as exercise increases in intensity the variability of the f_H diminishes. At the highest submaximal work load the cv of f_H is reduced to ~2%. This factor plays an important part in the improvement of the prediction of $\dot{V}O_{2\,max}$ when higher values of f_H (>165 beats/min) are used (Table 2). However, in statistical terms, when using the Åstrand-Rhyming nomogram in this way, the probability limits for a given predicted value approaching the "true" or observed $\dot{V}O_{2\,max}$ value are still large. This error can clearly be reduced further by either using two f_H values (Margaria) or the calculated regression line of $\dot{V}O_2$ on f_H (Maritz-Wyndham), but these methods still give errors of -529 ± 390 and -430 ± 356 ml, respectively, an improvement of only 2% on the Åstrand-Rhyming technique.

Unfortunately there is no physiologically valid solution to take account of the errors caused by random variation in f_H and $\dot{V}O_2$ measurements and nonlinearity in the $f_H/\dot{V}O_2$ curve, apart from fitting a precise curve to each individual subject's data. In practice this would be extremely difficult since it would involve repeated measurements of $\dot{V}O_2$ and f_H at all levels of exercise, and this is open to some criticism and drawbacks as the direct determination of $\dot{V}O_{2\,max}$. The only possible alternative would be to remove the "bias" in estimation by using the regression equation relating predicted and observed $\dot{V}O_{2\,max}$ shown in Fig. 7: $y = 932 + 0.828x$ where x = predicted $\dot{V}O_{2\,max}$ and y = observed $\dot{V}O_{2\,max}$. This equation has been applied to the present and previous data collected on a treadmill (11) and bicycle ergometer (10) from this laboratory and shown in Fig. 8. The method undoubtedly gives a more realistic estimation of $\dot{V}O_{2\,max}$ but the intersubject variability in the error due to the asymptotic nature of the $f_H/\dot{V}O_2$ curve (Table 2) limits the accuracy of the procedure for this population to ±15%. If work rate as an indirect measure of $\dot{V}O_2$ had been used, this error would probably have increased by ±6%.

Thus, even applying a large standard correction factor the method remains of limited value and provides only a crude guide to an individual's ability to perform at maximum effort; for accurate analysis of maximum aerobic power in man there appears to be no alternative but to measure $\dot{V}O_{2\,max}$ directly.

The study was undertaken partly to assess the work capacity of Royal Naval personnel. I am particularly indebted to Surg. Cdr. Mackay who organized the subjects and to J. Musgrove, A. V. Knibbs, and W. Tuxworth for invaluable technical assistance. I also thank Mr. M. Crowder for statistical advice and assistance.

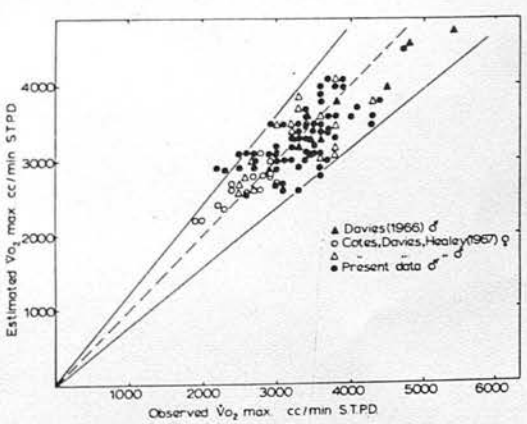


FIG. 8. Relationship of predicted $\dot{V}O_{2\,max}$ to observed $\dot{V}O_{2\,max}$ using the formula $y = 932 + 0.828x$, where x = predicted $\dot{V}O_{2\,max}$ and y = observed $\dot{V}O_{2\,max}$. ± 15% lines from the 45° line of identity are shown.

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Factors relating to the aerobic capacity of 46 healthy British males and females, ages 18 to 28 years

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(*The Medical Research Council's Pneumoconiosis and Environmental Physiology and Radiation Research Units* (1, 2 *respectively*), *Hampstead Laboratories* (3) *and Division of Computing and Statistics, Clinical Research Centre* (4), *University of London Institute of Child Health* (5).)

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1. Anthropometric and physiological data relevant to the capacity for exercise have been obtained in 46 healthy young adults, including 23 male and 20 female factory workers and three male athletes.

2. The measurements included maximal oxygen uptake on a cycle ergometer, cardiac frequency during submaximal exercise, total body potassium, the breadth of the limb muscles (at three sites), skinfold thickness at four sites, somatotype, forced vital capacity, transfer factor for the lung, total body haemoglobin, grip strength of the hand, vertical force during a standing jump and customary activity by questionnaire.

3. Linear regression relationships were obtained for: body potassium on thigh muscle, body weight and skinfold thickness (residual S.D. 6.4 g), maximal oxygen uptake on cardiac frequency and either body potassium or thigh muscle and forced vital capacity (residual S.D. 0.19 l min^{-1}), oxygen uptake during cycling on rate of work and body weight (residual S.D. 0.09 l min^{-1}) and maximal cardiac frequency on maximal oxygen uptake (residual S.D. 7.2 min^{-1}). The coefficient and constant terms for men and women, with the exception of the coefficient terms for the description of body potassium, do not differ to a material extent.

4. The use of these and other relationships provides a means for analysing the contributions to human exercise performance of body size, ethnic group and the effects of the environment.

Introduction

The aerobic capacity, the maximal consumption of oxygen during exercise, reflects the capacity for exercise of the subject under the conditions of the test. It is the resultant of the amount and effectiveness of the muscles which are used for the exercise and the capacity of the physiological processes whereby both oxygen is transported from the atmosphere to the muscles and heat is dispersed to the environment. The measurement is usually made over a number of periods of exercise, each of constant intensity and duration 4 to 6 min, separated by rest periods. The intensity is increased progressively from one period to the next, until the consumption of oxygen reaches a maximum. This procedure requires of the subject ample time, preferably over 2 or more days, a high degree of motivation and tolerance of discomfort, also freedom from coronary atheroma or other

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conditions which may affect health or performance during the test. To meet these requirements the measurement has traditionally been made on selected physically active subjects and has not been thought suitable for population studies. The alternative of using a battery of submaximal tests to assess attributes which underlie the capacity for exercise has not yet been validated for general use, though much work has been done along these lines (Von Döbeln, Åstrand & Bergström 1967; Ismail, Falls & MacLeod 1965; Holmgren & Åstrand 1966).

The present study assesses the feasibility of applying this method under survey conditions to untrained subjects, indicates which of the many possible indices of human structure and function are likely to be of practical use and attempts to establish what contribution, if any, the application of the method may make to the physiology of exercise. Preliminary findings have been communicated to the Physiological Society (Adamson & Cotes 1967; Burkinshaw, Cotes, Hughes & Jones 1967; Cotes, Davies & Healy 1967; Cotes, Davies & John 1967; Davies 1967).

Subjects and methods

Subjects

The subjects were 23 male and 20 female healthy factory workers and three male athletes of international standing; they volunteered to take part in the study after receiving a full description of what was entailed. Their selection is considered in greater detail below. The male factory workers were mainly doing fairly heavy work entailing the handling of metal castings; the females were mostly on light work in offices or laboratories, but the levels of their overall activity, including recreations, covered a wide range. Some details about the subjects are listed in table 3. They attended at the laboratory on 2 half days, with at

Table 1. Factors related to the capacity for exercise

Aspect	*Procedures and measurements*	
general	questionnaire	health, customary activity
body size and shape	anthropometry, photogrammetry	somatotype
body composition	skin fold thickness	
	roentgenogrammetry	limb muscle
	whole body counting	total body potassium
muscle force	static force	hand grip
	explosive force	standing jump
lung function	ventilatory capacity	forced expiratory volume and vital capacity
	gas transfer	transfer factor and residual volume
transfer function of blood	haemoglobin concentration	—
	total body haemoglobin	
response to exercise	submaximal exercise	cardiac frequency and ventilation
	maximal exercise	aerobic capacity

1 clear day between them; measurements were made on one attendance of unction, body haemoglobin and body potassium and on the other attendance dy dimensions including body anthropometry and roentgenogrammetry and es of performance during exercise. The procedures are summarized in table 1 given in greater detail opposite.

Medical assessment (*observer J. E. C.*)

ie medical history and present state of health including respiratory symptoms, cing habits and the level of customary daily activity were obtained by inter-, using as a basis the Medical Research Council's short questionnaire on res-ory symptoms (1960) with additional questions. The customary activity was equently graded on a 4-point scale.

Anthropometry (*observer P. R. M. J.*)

ne anthropometric measurements are listed in table 2. They were made using standard techniques which have been adopted for the International Biological gramme (Weiner 1969).

TABLE 2. ANTHROPOMETRIC AND SKINFOLD MEASUREMENTS

umferences	*skinfolds*	*skeletal measurements*	
eck	*biceps	chest depth	foot breadth
pper arm	*triceps	chest width	foot length
iest	*subscapular	biacromial	stature
iigh	*suprailiac	bi-iliac	sitting height
alf	calf	bicondylar humerus	subischial height
	juxta-nipple	bicondylar femur	(stature—sitting height)
		wrist breadth	suprasternal height
	*included in	hand breadth	anterior superior iliac spine height
	Σ4 skinfolds	ankle breadth	total arm length

Weight ratio index = ht $wt^{-\frac{1}{3}}$

Photogrammetry (*observer P. G. S.*)

Somatotype photographs were taken by the method of Jones & Stone (1964) ng the poses of Dupertuis & Tanner (1950), and a turntable of which the centre otation was 7.75 m from the nodal point of the camera lens. Enlargements were de on 30 cm × 25 cm waterproof paper, such that the 100 cm datum marks re reduced to 12 cm ± 0.04 cm (Tanner & Weiner 1949).

Anthroposcopic somatotyping (*observers P. R. M. J., P. G. S. and J. M. T.*)

This was done on a 13-point scale from inspection of the photographs after erence to standard somatotypes and the weight ratio index (table 2) (Sheldon, pertuis & McDermott 1954). For the male subjects the three observers rated prints independently; agreed ratings were then obtained from P. R. M. J. d P. G. S. and finally for all three observers. For the female subjects joint ratings

were obtained for two observers (P. R. M. J. and P. G. S.); these observers used the skinfold scores as an additional indication of the endomorphic component.

Radiographic determination of fat, muscle and bone (observers P. R. M. J. and P. C. R. H.)

Soft tissue radiographs were taken of the upper arm, thigh and calf on the left side of the body using the roentgenogrammetric techniques and poses developed at the Harpenden growth study (Tanner 1964). Fast films and regular intensifying screens were used and the X-ray set was fitted with a light-beam diaphragm and a 2 mm aluminium filter. The total radiation dose to the skin was less than 50 mrad (0.5 mJ kg^{-1}). The widths of the subcutaneous tissue, the muscle and the bone were taken on a line perpendicular to the long axis of the limb, or in the case of the humerus the long axis of the bone, with an X-ray caliper (Tanner & Whitehouse 1955); the measurements were made at the mid shaft of the humerus, the maximum diameter of the calf and a point on the thigh one-third of the subischial height measured up from the distal border of the femoral condyles.

Body potassium (observers L. B. and D. W. K.)

The body potassium is intracellular in its distribution and as such provides an index of body muscle. The technique used for its estimation makes use of the fact that natural potassium contains a constant proportion of the long-lived radioactive isotope, ^{40}K. This isotope emits penetrating γ-rays, some of which can be detected by radiation counters placed around the body. The observed counting-rate is a measure of the amount of potassium present.

The whole-body counter used in this work comprises three large plastic scintillation detectors grouped around a chair. The apparatus is housed in a room with walls of steel, 13 cm thick, lined with 2.5 cm of lead (Burkinshaw & Spiers 1967). Each subject was measured in the counter for 20 min, and the observed gross counting-rate was corrected for background, recorded for 60 min. Whole-body potassium was calculated by multiplying the net counting-rate by a calibration factor, which is a linear function of the ratio of the subject's weight and height. This calibration had been established previously for 21 men and 17 women, by a technique in which each subject was given a calibrated dose of the short-lived radioisotope ^{42}K (Burkinshaw 1967). The coefficient of variation of the potassium estimates reported in this paper is about 4%.

Muscle strength (observer G.T.A.)

The grip strength was measured with a Collins-type spring steel hand dynamometer. The force during a standing jump was assessed in terms of the peak vertical component recorded from a force platform.

The procedures for both measurements were explained and demonstrated to the subject; then, for the hand grip, the subject made three contractions using the dominant hand. The mean of these was calculated and used as an index of static force. For the standing jump the subject performed the manoeuvre three

times, but in some instances preceded the jump by a bounce which invalidated the measurement; the better of the first two results which were technically satisfactory was used as an index of explosive force.

Lung function (observers M. J. S., A. M. H. and M. R.)

The ventilatory capacity was measured as the forced expiratory volume ($FEV_{1.0}$) and forced vital capacity with a waterless bellows spirometer, fitted with an electronic timing device (Collins, McDermott & McDermott 1964); the values for each subject were obtained from the mean of three technically satisfactory expirations after two practice attempts. The transfer factor (diffusing capacity) for the lung was measured by the single breath carbon monoxide method. For this test the volume of the breath, the time of breath holding and the volume, size and position of the gas sample for analysis were controlled by an automatic apparatus (Meade, Saunders, Hyett, Reynolds, Pearl & Cotes 1965). Unfortunately, in transit, the sensing device on the spirometer head developed an alinement fault which had the effect of cutting short the test inspiration. As a result the values reported for the transfer factor in the male and female subjects are, on average, too low by about 20 and 10 % respectively. The lung volume used for the calculation was that obtained from the dilution in the lung of the single breath of test gas; the volume comprised the volume inspired and the estimated residual volume. The diffusing capacity of the alveolar capillary membrane and the volume of blood in the alveolar capillaries were obtained by the method of Roughton & Forster (1957) from measurements of the transfer factor at two levels of oxygen tension. For this purpose haemoglobin concentration was obtained on a sample of blood from the lobe of the ear by use of an E.E.L. electrophotometer (cyanmethaemoglobin method). Further details of the techniques are available elsewhere (Cotes 1968).

The total body haemoglobin was estimated by a modified carbon monoxide method from the change in the tension of carbon monoxide in a closed breathing circuit containing oxygen, following the administration to the subject of a known amount of carbon monoxide. The tensions are converted to saturations by use of the Haldane equation: this requires the tension of carbon monoxide and the saturation and tension of oxygen in the alveolar capillaries. The tension of carbon monoxide is the same as that in the apparatus, provided that sufficient time (usually 15 min) is allowed for equilibration. The saturation of oxygen (S_{O_2}) is equal to ($100 - S_{CO}$) since the subjects are breathing oxygen-enriched gas. The tension of oxygen is not measured directly: instead, the tension of carbon monoxide (P_{CO}) in the apparatus is measured at two tensions of oxygen (P_{O_2} (i) and P_{O_2} (ii)), where the second is obtained by the addition of nitrogen to the apparatus after equilibration with carbon monoxide. In these circumstances the Haldane equation for calculation of the saturation of carbon monoxide (S_{CO}) has the form

$$\frac{S_{CO}}{230\ (100-S_{CO})} = \frac{P_{CO}\,(\text{i}) - P_{CO}\,(\text{ii})}{P_{O_2}\,(\text{i}) - P_{O_2}\,(\text{ii})}$$

The total body haemoglobin (*THb*) is then obtained as follows:

$$THb\ (\text{g}) = \frac{\text{CO absorbed (ml STPD)} \times 100}{1.34 \times \Delta S_{CO}\ (\%)}$$

where $S_{CO}(\%)$ is the increase in the saturation of haemoglobin with carbon monoxide over the period of rebreathing.

The initial saturation at the start of the test is the resultant of endogenous production, carbon monoxide absorbed during measurement of the transfer factor and exogenous carbon monoxide from other sources including cigarettes. For application of the method under survey conditions the subjects were asked not to smoke on the day of the test. In these circumstances the average initial saturation based on a separate study of 37 subjects is 1.3 % (S.D. 0.31 %); this value was assumed for the majority of subjects who did not smoke on the day of the test. An initial saturation of 2.15 % was assumed for the remainder.

Response to exercise (*observers C. T. M. D., W. W., G. E. C. C. and P. G.*)

The exercise was performed on a cycle ergometer (Müller) in sessions which lasted 90 min for subjects studied in pairs. During a preliminary period electrodes were applied and the subjects were allowed to become accustomed to the procedure. For this purpose they undertook a 4 min warm-up period of exercise at 450 kp m min^{-1}: this was followed by a rest period of 10 min. The first period of test exercise was for 6 min at a work level of 450 kp* m min^{-1} (73.5 W); it was followed by a rest period of 8 min or until the cardiac frequency had fallen to within 5 beats per min of the frequency before the start of exercise. The subject then cycled for a further 6 min at a higher work level; this was for the male subjects 900 kp m min^{-1} (147 W) and for the female subjects 720 or, in two instances, 540 kp m min^{-1} (118 and 88 W respectively). After a further rest period the maximal oxygen intake was assessed by means of a continuous work procedure after the method of Binkhorst & Van Leeuwen (1963). For this purpose the work level was increased at 2 min intervals from a starting-point which was determined in the light of the response of the subject to submaximal exercise. The maximum was judged to have been achieved when an increase in work load caused no material change in the consumption of oxygen (i.e. at least two values agreeing to within $\pm 5\%$). The cardiac frequency during the last 2 min of exercise was used as a guide to maximal effort, but not as a criterion of maximal oxygen uptake.

The subjects inhaled through a low resistance valve (Bannister & Cormack 1954) and the expired gas was collected via a short length of smooth tubing (internal diameter 2.8 cm) in a Douglas bag which was suspended to reduce its resistance. The bag was subsequently emptied through a dry gas meter (Parkinson and Cowan Ltd). The cardiac frequency was recorded continuously through light-weight clip electrodes (Davies & Copland 1964) in conjunction with an E.C.G. differential amplifier and a linear instantaneous rate meter (Nielson 1963). The respiratory

* 1 kp (kgf) $\approx$ 9.8 N.

frequency was monitored from a bead thermistor placed in the mouthpiece (Davies & Nielson 1963). The consumption of oxygen during submaximal exercise was determined over the 4th to 6th min and at maximal effort during the final minute at each work load. The gas from the Douglas bags was analysed for oxygen with a paramagnetic meter (Servomex Ltd) and for carbon dioxide with a katharometer (Cambridge). Details of the accuracy and reliability to be expected with these methods have already been given (Cotes & Woolmer 1962; Davies & Shirling 1967). The frequency of pedalling was for the male subjects 60 min^{-1} and for the females 50 min^{-1}.

The relationships of ventilation and cardiac frequency to oxygen uptake at the two levels of submaximal exercise were used to obtain by interpolation the ventilation and cardiac frequency at an oxygen uptake of 1.5 l min^{-1} (cf. I.L.O. 1966). The relationship for cardiac frequency was also extrapolated linearly to an estimated maximal frequency of 195 min^{-1} and the corresponding oxygen uptake noted; this was used as an estimate of the maximal oxygen uptake.

Administration, selection of subjects and treatment of data
(J. E. C., M. J. R. H., G. B., C. J. J. and E. D.)

The choice of Leeds for the survey was determined by the facilities for measurement of whole-body potassium; the remaining measurements were made in the University Department of Physical Education, where 186 m^2 (2000 ft^2) of laboratory space were made available for the purpose. The space was allocated as follows: exercise studies 56 m^2 (600 ft^2), anthropometry, 84 m^2 (900 ft^2), lung function and haemoglobin, 46 m^2 (500 ft^2). The measurements were made over a period of 2 weeks in early summer (mean temperature 20 °C), mainly by visiting staff from other centres. In all 17 scientists and technicians took part, of whom 14 where whole-time and three part-time. It was planned to study 24 working male and 24 female subjects in a 5-year age group somewhere in the age range 20 to 30 years, also as many athletes of international status as could be assembled in the time. Introductions to the subjects were obtained through the Medical Officer of Health for Leeds and the Personnel and Medical Departments of two large industrial concerns; approximately 50 % of those who were approached agreed to take part. The subjects comprised 20 male and 20 female factory workers and 6 male athletes. Three of the athletes were of international status; the other three were college athletes whose data for all the indices which were examined fell within the ranges for the other male subjects. These two groups of subjects were subsequently combined. Some measurements which were rejected on technical grounds were repeated including maximal exercise (5 subjects), photogrammetry (8 subjects), roentgenogrammetry (1 limb in each of 3 subjects), certain body measurements (1 subject) and total body haemoglobin (3 subjects). In addition, missing values were estimated for the oxygen uptake or the cardiac frequency during one period of exercise in each of three subjects, and of the calf width in another; the estimates were based in the former subjects on the regression of cardiac frequency on oxygen

uptake and in the latter on the regression of calf muscle width on calf circumference. Measurements of muscle strength on 16 subjects were obtained at the place of employment after the completion of the main survey. By these means the number of missing readings was reduced to seven, including five measurements of explosive force and two of the cardiac frequency during maximal exercise.

The analysis of the data was undertaken in stages, including an extensive graphical study and the construction of a cross-correlation matrix for 20 variables (table 4); this formed the basis for detailed treatment of individual indices.

Results

The mean values and ranges in the factory workers and the individual values in the athletes for 29 of the indices are listed in table 3; the corresponding correlation coefficients for 20 of the indices describing the male and female factory workers are included in table 4. The coefficients for the logarithms are essentially similar. From this treatment of the data, 11 indices were selected for more detailed analysis of their relation to the maximal oxygen uptake. These are indicated in table 4 and are considered in relation to each other and to the maximal oxygen uptake in the following paragraphs. They were chosen as describing, so far as was practicable, the dimensions and function of the several components of the oxygen transport system and body muscle, with a bias towards indices which were appropriate for surveys; thus as well as limb muscle and body potassium, the skinfold thickness was included in the expectation that when combined with body weight it might provide a more convenient index of body muscle. For the same reason the cardiac frequency at the constant work rate of 450 kp m min^{-1} (73.5 W), because it is easier to attain than the cardiac frequency at an oxygen uptake of 1.5 l min^{-1}, was also included in the analysis, despite its lower correlation with maximal oxygen uptake. Some variables were included because of their relevance for oxygen transport, despite their not yielding significant correlation coefficients for both sexes separately in the present study; the indices of lung function and total haemoglobin fall into this category. Other variables which exhibited comparable or lesser degrees of correlation with maximal oxygen uptake were excluded on account of their being less directly connected with the oxygen transport-muscle system, for example additional indices of body size, somatotype, grip strength, performance on a force platform and customary activity assessed by questionnaire.

Assessment of body muscle

Skeletal muscle was assessed indirectly for the body as a whole, by measurement of the total body potassium and directly for the three limb sites, by soft tissue roentgenogrammetry. The sum of the muscle widths for the thigh, upper arm and calf was included in the main analysis (table 4); the index proved to be highly correlated with the total body potassium and indices of performance during exercise. Subsequent analysis showed that of the three components, the thigh muscle which comprised, on average, 46 % of the total width, also contributed

TABLE 3. MEAN VALUES AND RANGES FOR SOME

index number	units	factory workers: male 23		factory workers: female 20		male athletes: A	male athletes: B	male athletes: C
age	a	25.0	18–28	23.7	20–28	27.5	25.3	22.8
height	m	1.76	1.61–1.91	1.62	1.5–1.8	1.84	1.72	1.84
weight	kg	71.4	56–92	55.0	47–69	106.1	74.6	94.0
blood haemoglobin (Hb%)	g%	14.8	13.3–16.9	13.3	11.7–14.6	107	104	110
Σ 4 skin-fold thicknesses	cm	3.12	1.68–7.94	4.90	3.37–11.4	3.17	1.81	5.62
Σ 3 muscle widths	cm	28.1	23.1–32.7	23.1	20.5–26.3	38.1	32.3	33.4
thigh muscle width	cm	13.0	11.5–14.9	10.5	8.65–12.0	17.8	14.2	15.3
whole body potassium (K)	g	144	115–180	91.1	78–109	250	188	175
total body haemoglobin (HbT)	g	776	576–1094	510	353–741	1080	1168	710
level of activity	—	2.3	1–3	1.5	1.0–2.5	4	4	4
forced expiratory volume ($FEV_{1.0}$)	l	4.44	3.25–5.48	2.97	2.3–3.9	5.30	4.96	5.15
forced vital capacity (FVC)	l	5.66	4.38–7.72	3.66	2.9–4.0	6.38	6.34	6.47
estimated residual volume (ER)	l	1.41	0.75–1.99	1.38	0.5–2.3	1.65	2.27	1.76
transfer factor* (TF)	ml min^{-1} $Torr^{-1}$	30.0	21.3–43.8	23.4	17.0–30.3	45.0	42.1	40.4
diffusing capacity of alveolar membrane (Dm)	ml min^{-1} $Torr^{-1}$	47.6	105–24	37.0	53–27	61.8	52.9	67.4
volume of blood in alveolar caps (Vc)	ml	63.7	101–34	55.2	95–31	132.6	174.1	85.4
exercise at 450 kp m min^{-1}								
oxygen consumption ($\dot{V}_{O_2\,450}$)	l min^{-1}	1.27	1.05–1.43	1.11	0.9–1.3	1.48	1.40	1.37
cardiac frequency (CF_{450})	min^{-1}	116	88–147	144	109–178	97	115	108
ventilation ($\dot{V}_{e\,450}$)	l min^{-1}	30.9	23.6–48.9	29.1	20.3–36.8	29.0	32.5	23.3
cardiac fr. at $\dot{V}_{O_2}$ 1.5 l min^{-1} ($CF_{1.5}$)	min^{-1}	125	94–152	169††‡	131–192	98	118	111
ventilation at $\dot{V}_{O_2}$ 1.5 l min^{-1}	l min^{-1}	36.5	29.2–52.3	45.4††‡	36.0–64.1	29.4	34.8	25.5
maximal exercise								
oxygen consumption ($\dot{V}_{O_2max}$)	l min^{-1}	3.4	2.7–4.7	2.2	1.6–3.0	5.37	3.81	4.56
cardiac frequency	min^{-1}	190§	172–206	193†	180–204	187	181	197
$\dot{V}_{O_2\,max}$ per kg body weight	ml min^{-1} kg^{-1}	48.5	37–67	39.2	29–53	50.7	51.0	48.5
ventilation ($\dot{V}_{e\,max}$)	l min^{-1}	119.2	66–169	79.5	51–107	154.4	138.5	147.6
respiratory frequency	min^{-1}	48.7	32–72	42.9	30–58	49	51	56
$\dot{V}_{e\,max}$/MBC	%	79.4	56–104	74.8	47–103	80.4	77.4	79.3
Vt/FVC	%	47.8	35–76	52.4	37–75	49.4	42.8	40.7
hand grip	kp	44.4	36–55	24.9	17–33	59.9	66.7	59.1
standing jump, peak force	kp	172†	129–223	132†	105–175	282	251	252

* Results subject to systematic error (see methods). † nineteen subjects. ‡ at $\dot{V}_{O_2}$ 1.0 139 (111–173)/min and 24.0 (16.5–29.5) l min^{-1}. § twenty-two subjects.
A, B and C, for identity see text.

TABLE 4. CROSS CORRELATION MATRIX FOR 20 INDICES

Correlation coefficients which are significant at or below the 5 % level are shown in heavy type.

	ht	wt	Hb %	$FEV_{1.0}$	FVC	ER	TF	$D\,m^{-1}$	$V\,c^{-1}$	HbT	$CF_{1.5}$	$\dot{V}_{O_2\,450}$	$\dot{V}_{O_2\,max}$	Σ4 skinfolds	Σ3 muscles	grip	*K*	CF_{450}	$\dot{V}_{c\,450}$	level of activity
*ht		**0.45**	0.12	**0.50**	**0.60**	**0.56**	**0.53**	**−0.60**	0.04	0.26	−0.39	0.15	0.26	−0.28	0.02	0.14	0.39	−0.25	0.04	**−0.46**
*wt	0.03		0.25	**0.48**	**0.63**	**0.45**	0.34	−0.28	−0.03	**0.43**	−0.18	**0.78**	0.37	**0.47**	**0.54**	−0.02	**0.73**	0.04	0.35	−0.22
Hb %	0.03	0.09		0.27	0.41	0.27	0.27	0.00	−0.09	−0.09	0.27	0.06	−0.04	0.10	0.16	0.02	0.17	0.28	0.05	−0.28
$FEV_{1.0}$	0.23	−0.20	−0.37		**0.84**	**0.74**	**0.62**	−0.48	−0.24	0.11	−0.15	0.17	0.41	−0.19	0.34	−0.25	**0.51**	−0.09	0.15	0.04
*FVC	0.17	−0.01	−0.27	**0.82**		**0.85**	**0.70**	**−0.58**	−0.24	0.30	−0.20	0.30	**0.53**	−0.20	**0.45**	0.03	**0.65**	−0.08	0.22	−0.26
ER	0.37	−0.39	−0.11	0.03	−0.04		**0.79**	**−0.70**	−0.38	0.20	−0.30	0.14	0.40	−0.27	0.32	0.13	**0.52**	−0.20	0.01	−0.29
*TF	−0.16	−0.44	0.11	−0.26	−0.14	**0.63**		**−0.72**	**−0.56**	0.33	−0.14	0.06	0.32	−0.24	0.24	0.23	**0.54**	−0.06	−0.03	−0.41
$D\,m^{-1}$	0.04	**0.56**	0.10	−0.12	−0.21	**−0.59**	**−0.80**		0.02	−0.03	0.22	−0.01	−0.23	0.28	−0.07	−0.08	−0.28	0.17	−0.01	0.24
$V\,c^{-1}$	0.06	0.06	−0.11	0.39	0.36	**−0.47**	**−0.66**	0.16		−0.40	0.08	0.04	−0.35	0.12	−0.21	−0.23	−0.34	0.03	0.03	0.11
*HbT	−0.17	**0.49**	−0.24	0.17	0.28	−0.07	−0.07	0.15	−0.15		−0.24	0.38	**0.45**	0.01	**0.44**	0.31	**0.66**	−0.12	0.00	−0.17
* $CF_{1.5}$	0.11	−0.40	−0.43	−0.04	−0.24	**0.48**	0.28	−0.25	−0.34	−0.22		−0.13	**−0.58**	0.26	−0.30	−0.05	−0.40	**0.95**	0.24	0.14
$\dot{V}_{O_2\,450}$	−0.29	0.18	−0.14	0.12	−0.02	**−0.49**	−0.43	0.32	0.32	−0.00	−0.03		0.25	**0.47**	0.36	0.00	**0.59**	0.11	**0.56**	−0.02
* $\dot{V}_{O_2\,max}$	−0.17	0.35	0.40	0.05	0.32	**−0.52**	−0.21	0.13	0.34	0.23	**−0.87**	−0.06		−0.26	**0.57**	−0.02	**0.69**	**−0.52**	−0.14	0.05
*Σ4 skinfolds	−0.21	**0.83**	−0.02	−0.31	−0.25	−0.35	−0.34	**0.56**	−0.11	0.27	−0.06	0.26	0.02		0.05	−0.09	−0.03	0.38	0.30	−0.04
*Σ3 muscles	**−0.46**	**0.53**	0.28	−0.10	0.16	**−0.46**	−0.05	0.10	0.14	0.36	**−0.79**	0.09	**0.78**	0.35		−0.07	**0.72**	−0.29	−0.04	−0.05
Grip	−0.03	0.22	0.15	−0.01	0.20	−0.15	0.00	−0.08	0.14	−0.03	0.21	0.09	0.03	0.24	0.01		0.13	0.02	−0.00	−0.15
*K	0.04	0.35	0.12	0.22	**0.49**	−0.41	−0.35	0.14	**0.48**	0.36	**−0.77**	−0.02	**0.77**	−0.08	**0.58**	0.12		−0.25	0.06	−0.10
* CF_{450}	0.16	−0.12	−0.35	−0.26	−0.35	0.31	0.10	−0.11	−0.19	−0.10	**0.84**	−0.05	**−0.70**	0.08	**−0.70**	0.19	**−0.60**		0.37	0.05
$\dot{V}_{e\ 450}$	0.06	−0.08	−0.25	0.44	0.24	0.07	−0.34	0.05	**0.49**	−0.09	0.10	0.36	−0.17	−0.11	−0.17	−0.10	−0.15	0.14		0.04
level of activity	−0.06	0.10	0.18	−0.05	0.10	−0.39	−0.11	0.13	0.18	0.19	**−0.70**	−0.27	**0.64**	−0.20	**0.47**	−0.42	**0.51**	**−0.46**	−0.10	

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For explanation of abbreviations and symbols see table 3. Indices selected for detailed analysis are indicated * (for details see text).

ost to the overall relationships. On this account, for most purposes, it has been ssible to omit the arm and calf measurements without loss of accuracy. However, e latter measurements had the advantage in the present study of being more adily predicted from the other data. This was due to the arm and calf circum- ences and skinfold measurements being highly correlated with the corresponding easurements obtained off the soft tissue X-rays at the same site. By contrast for ie thigh, where no measurement was made of the skinfold thickness, the sum of ie four skinfolds obtained elsewhere in the body (see Methods) proved not to be satisfactory alternative.

Assessment of total body potassium

The total body potassium was found to be highly correlated with the maximal xygen uptake and indices related to it. Amongst the individual muscle measure- ients the correlation with potassium for the thigh muscle (figure 1) was rather higher ian for the arm muscle and at least as high as for the sum of the arm, thigh and calf ieasurements combined. The correlation was marginally improved by converting he muscle width into a quasi-volume by multiplying width squared by standing eight. A further increase in precision was obtained by calculating the regression f potassium on this estimate of total muscle, together with the body weight and he product (sum of 4 skinfolds) × (height) × (biacromial diameter). The product is measure of total fat and in combination with weight gives a measure of lean ody mass. The best guide, however, for both sexes, was obtained from straight- orward linear regressions on weight (wt), the width of the thigh muscle (m) and he sum of the 4 skinfold thicknesses (f) as follows:

$$\text{for men } K\text{ (g)} = 5.6\,m\text{ (cm)} + 1.19\,wt\text{ (kg)} - 4.4\,f\text{ (cm)} \qquad (1a)$$

$$\text{for women } K\text{ (g)} = 4.0\,m\text{ (cm)} + 1.17\,wt\text{ (kg)} - 3.2\,f\text{ (cm)} \qquad (1b)$$

The standard deviations about these regression lines are respectively 7.4 and 4.7 g; the corresponding coefficients of variation, on account of differences in mean values for body potassium between the sexes, are the same at 5.2 %.

Role of somatotype

For the male subjects the ratings for ectomorphy, mesomorphy and endomorphy were found to be appropriately correlated with the weight and height, the measure- ments of skinfold thickness and limb muscle, the body potassium and the aerobic capacity (table 5). For the female subjects the factors which reflected the ratings for ectomorphy and mesomorphy were similar to those for the males, but the rating for endomorphy was inappropriate in that it was not independent of limb muscle.

Lung function and respiratory symptoms

The subjects included in the analysis were free from persistent cough and sputum, but a proportion of them did have minor symptoms including occasional wheeze and morning phlegm. Three-quarters of the subjects were cigarette smokers, in- cluding 10 who smoked more than 15 cigarettes per day; many of the respiratory

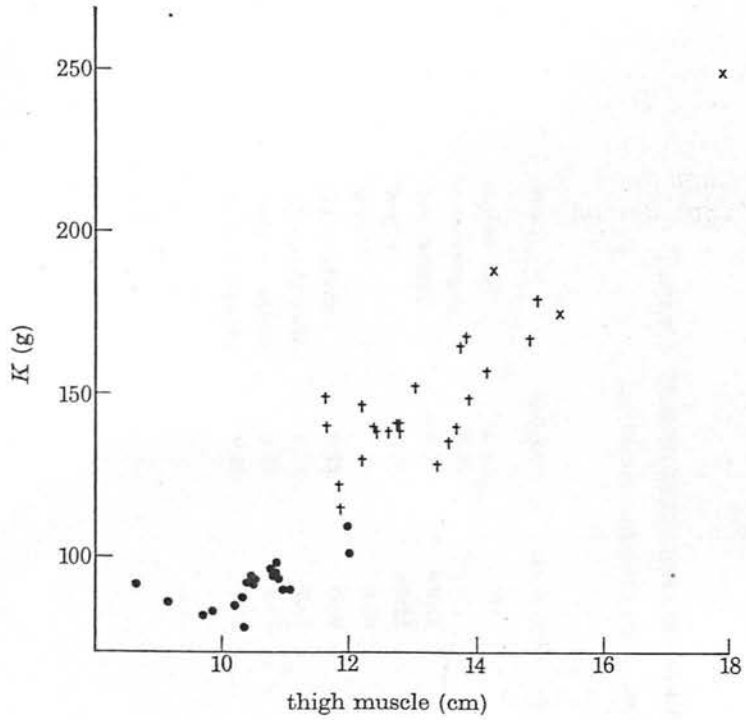

Figure 1. Relationship of body potassium (K) to thigh muscle width. (For key see below.)

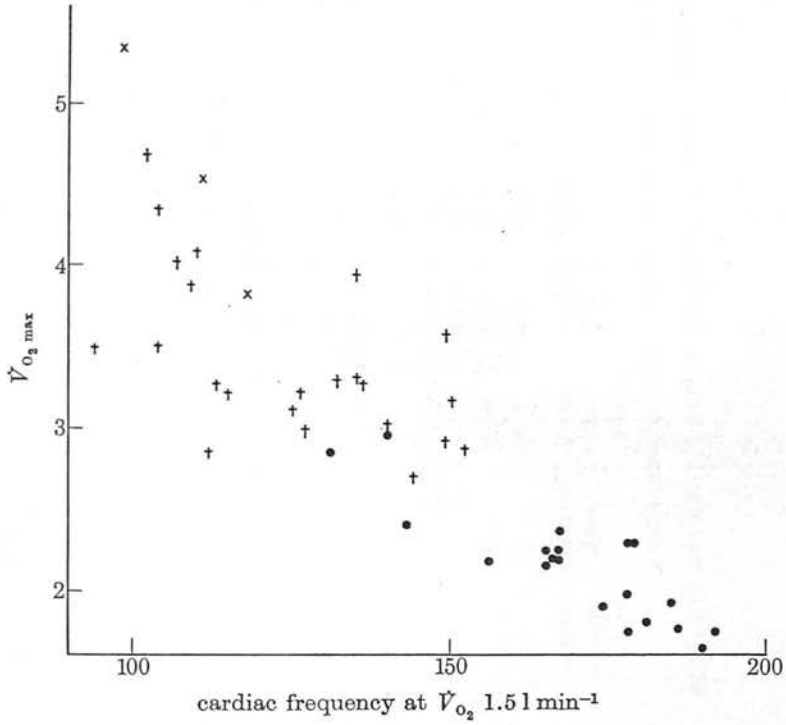

Figure 2. Relationship of maximal oxygen uptake ($\dot{V}_{O_2\,max}$) to cardiac frequency at an oxygen uptake ($\dot{V}_{O_2}$) of 1.5 l/min.

Key to figures 1 *and* 2

† male
● female
} factory workers × male athletes

TABLE 5. SOMATOTYPE, RELATIONSHIP TO OTHER VARIABLES IN 23 MALE AND 20 FEMALE SUBJECTS

Correlation coefficients which are significant at or below the 5% level are shown in heavy type.

	endomorphy	mesomorphy	ectomorphy	height	weight	Σ4 skinfolds	Σ3 muscle widths	body potassium	aerobic capacity
endomorphy		0.28	**−0.80**	−0.31	**0.59**	**0.88**	0.26	0.13	−0.18
mesomorphy	0.34		**−0.62**	0.34	0.31	0.05	**0.75**	**0.60**	**0.44**
ectomorphy	**−0.86**	−0.41		**0.57**	**−0.44**	**−0.63**	−0.42	−0.27	−0.03
height	−0.38	−0.22	**0.67**	see table 4					
weight	**0.80**	0.34	**−0.67**						
Σ4 skinfolds	**0.83**	0.09	**−0.69**						
Σ3 muscle widths	**0.53**	**0.60**	**−0.69**						
body potassium	0.22	**0.59**	−0.27						
aerobic capacity	0.26	**0.51**	**−0.47**						

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symptoms were probably attributable to this cause. However, the total numbers of subjects were too small for a satisfactory analysis of the contribution of smoking to the overall result. The findings on assessment of lung function (table 3) were essentially those to be expected for subjects of this age and size living in an urban environment. The indices exhibited a high degree of inter-correlation (table 4); it was, therefore, practicable to select one index as representative of this group of tests for inclusion in the final analysis. The vital capacity, on account of its simplicity and wide availability, was chosen for this purpose.

The vital capacity and its inspiratory component under quiet resting conditions (inspiratory capacity) were compared with the tidal volume during maximal exercise. The maximal tidal volume was less than the inspiratory capacity in all but three instances; it was, on average, half the vital capacity (table 3).

The ventilatory capacity expressed as the indirect maximum breathing capacity (i.e. 36.8 $FEV_{1.0} - 3.8$, McKerrow, McDermott & Gilson 1960) was similarly compared with the maximal exercise ventilation. On average the proportion of the maximum breathing capacity used during exercise ($\dot{V}_{e\ max}$/MBC) was just under 80 % for all groups of subjects (table 3).

The transfer factor (diffusing capacity) for the lungs proved not to be correlated with the maximum oxygen uptake; however, the volumes of test gas inhaled during the measurement were, on average, less than those of which the subjects were capable (see Methods). They were subsequently adjusted on the basis that within subjects the transfer factor varies, on average, by 3 ml min^{-1} $Torr^{-1}$ l^{-1} of inspired gas (Cotes 1968). The use of these revised values did not, however, improve the relationship.

Total body haemoglobin

The total body haemoglobin was positively correlated with body weight and, in the male subjects, with total body potassium, limb muscle and maximal oxygen uptake. In the female subjects the correlation coefficients for total haemoglobin with the first two but not the third of these variables also approached the 5 % level of significance.

In relation to this result the use of an average value for the initial saturation of haemoglobin with carbon monoxide (see Methods) may have increased the variability of the data. But the effect is likely to have been small. Any errors are related to the amount of haemoglobin available to take up the test dose of carbon monoxide and are therefore larger for the data for the male than for the female subjects. They do not explain the lower correlation coefficients in the latter, for which there must be some other explanation.

Muscle strength

The measurements of grip strength for the dominant hand were included in the main analysis (table 4); they were also examined with respect to other indices including the width of the muscle in the left upper arm, the width of the left hand and the rating for mesomorphy. Although the mean grip strength varied between

the different groups of subjects in a predictable fashion (table 3), within the groups it was unrelated to any of the indices with which it was compared.

The explosive force during a standing jump, on account of technical failures, was not available for some of the subjects, whilst in others only one technically satisfactory measurement was available; for these reasons it was not included in the main analysis. Instead, it was analysed separately with respect to the maximal oxygen uptake, the grip strength, the thigh muscle and the total body potassium. In the male subjects, but not in the female subjects, the explosive force proved to be positively correlated with the latter two of these variables (correlation coefficients for the thigh muscle, 0.64 and for the body potassium, 0.48). All the relationships were significant when combined ignoring sex.

Submaximal exercise

The oxygen uptakes during submaximal exercise were linearly related to the rate of work against the ergometer and, in the male subjects, to body weight. Combination of these two relationships yielded the following equation:

$$\dot{V}_{O_2} = 1.93W + 6.84wt - 94 \text{ (S.D.90) ml min}^{-1} \quad (2)$$

where $\dot{V}_{O_2}$ is oxygen uptake in ml min^{-1}, W is the rate of work on the ergometer in kp m min^{-1} and wt is body weight in kg. For the female subjects the partial regression coefficient of oxygen uptake on work rate was nearly identical with that for males of the same average weight (55.7 kg); the regression coefficient on body weight was effectively zero, but the range of weights was relatively small.

TABLE 6. PREDICTION OF MAXIMAL OXYGEN UPTAKE IN FACTORY WORKERS

Mean values in males 3.43 l min^{-1} and in females 2.14 l min^{-1}

	coefficient of variation	
basis for prediction	males	females
mean for the group	14.1	15.6
extrapolation from submaximal exercise	10.0	6.5
body potassium (K)	10.7	10.9
cardiac frequency at $\dot{V}_{O_2}$ 1.5 l min^{-1} ($CF_{1.5}$)	11.7	8.2
K and $CF_{1.5}$ combined	9.5	8.1
Σ 3 muscle widths	11.9	10.0
thigh muscle	11.4	11.2
forced vital capacity (FVC)	12.1	15.3
$CF_{1.5}$ Σ 3 muscle widths and FVC	9.7	7.9
$CF_{1.5}$, thigh muscle and FVC	9.7	7.4*
$CF_{1.5}$, FVC, height and total haemoglobin	10.1	—

* or using $CF_{1.0}$ 8.6

The cardiac frequencies at the constant work rate of 450 kp m min^{-1} and at the constant oxygen uptake of 1.5 l min^{-1} were examined in relation to the other variables (table 4). The frequencies were highly correlated with each other and

both were negatively correlated with the maximal oxygen uptake, the frequency at an oxygen uptake of 1.5 l min^{-1} being the better predictor in this respect (figure 2). In the female, but not in the male subjects, both indices were also correlated with the body potassium and with estimates of body muscle.

Maximal exercise

Mean values for the oxygen uptake and the cardiac frequency during maximal exercise are listed in table 3; the exercise ventilation and the tidal volume are considered in relation to the ventilatory capacity in the paragraph on lung function.

The cardiac frequency during maximal exercise for all the subjects together was negatively correlated with the maximal oxygen uptake; the relationship was

$$CF_{max}, \text{ min}^{-1} = 203 - 4.23\ \dot{V}_{O_2\,max}\ (\text{l min}^{-1}), \text{ S.D. } 7.2. \quad (3)$$

However, despite this association, the maximal oxygen uptakes derived by extrapolation of the data for maximal exercise to an assumed maximal frequency of 195/min (see Methods) were highly correlated with the measured values. For both male and female subjects the predictions were too low by about 0.3 l, although in the case of the females the cardiac frequencies at the higher of the two levels of submaximal exercise were close to the assumed maximal value. The variability of prediction of the maximal oxygen uptake using this method is listed in table 6, which also includes the variabilities using the other factors related to the capacity for exercise which are discussed above. The coefficients of variation which are quoted are the residual standard deviations about the regression lines expressed as percentages of the mean values. As in the case of the prediction of body potassium, the regression relationships which include limb muscle have been calculated for the arm, thigh and calf muscle separately and in combination. The inclusion of the width of the arm muscle does not materially reduce the overall variability, whilst the reduction using the thigh muscle alone is as great as for the three muscle widths combined. The results suggest that as an alternative to the extrapolation procedure, the maximal oxygen uptake may be predicted with similar precision from the linear regressions on the cardiac frequency at an oxygen uptake of 1.5 (or 1.0) l min^{-1}, together with either the body potassium, the forced vital capacity and the width of the muscle mass in the thigh. For both the male and female factory workers the following relationships obtain:

$$\dot{V}_{O_2\,max} = 0.015\ K\ (\text{g}) - 0.0114 CF_{1.5} + 2.70\ (\text{S.D. } 0.23)\ \text{l min}^{-1} \quad (4)$$

$$\dot{V}_{O_2\,max} = 0.19\ m\ (\text{cm}) + 0.16 FVC\ (\text{l}) - 0.011 CF_{1.5} + 1.38\ (\text{S.D.}0.26)\ \text{l min}^{-1}. \quad (5)$$

where $\dot{V}_{O_2\,max}$ is the maximal oxygen uptake, $CF_{1.5}$ is the cardiac frequency at an oxygen uptake of 1.5 l min^{-1}, K is the total body potassium, FVC is the forced vital capacity and m is the width of the thigh muscle. For the female subjects the latter relationship has also been calculated using the cardiac frequency at an oxygen uptake of 1.0 l min^{-1} ($CF_{1.0}$); then

$$\dot{V}_{O_2\,max} = 0.19m\ (\text{cm}) + 0.094 FVC\ (\text{l}) - 0.0099 CF_{1.0} + 1.19\ (\text{S.D.}0.19)\ \text{l min}^{-1} \quad (6)$$

For the male subjects the inclusion of an estimate of total body haemoglobin also improves the relationship (table 6).

TABLE 7. PHYSIOLOGICAL VARIABLES IN 3 ATHLETES. COMPARISON OF OBSERVED VALUES WITH PREDICTIONS BASED ON DATA FOR FACTORY WORKERS

variable	basis for prediction	prediction equation	observed	predicted	difference
$\dot{V}_{O_2,\,max}$ l min^{-1}	K, $CF_{1.5}$	4	4.58	4.52	+0.06
$\dot{V}_{O_2,\,max}$ l min^{-1}	m, FVC, $CF_{1.5}$	5	4.58	4.23	+0.35
K g	wt, m, f	1	204	182	+23*
$\dot{V}_{O_2\,450}$ l min^{-1}	W, wt	2	1.42	1.40	−0.02
$\dot{V}_{O_2\,900}$ l min^{-1}	W, wt	2	2.16	2.27	−0.11
FVC	age, ht	†	6.4	5.3	+1.1*

*$p < 0.05$. † See Cotes 1968.

Athletes

The results for the three athletes are listed individually in table 3, where the columns refer respectively to the shot and discus thrower (A), the weight lifter (B) and the oarsman (C); the first in particular is exceptionally powerfully built. Their muscle measurements are mostly outside the range for the other male subjects and the values obtained for indices of lung function, muscle strength, cardiac frequency during submaximal exercise and maximal oxygen uptake also suggest that their capacity for exercise is greater. The results of applying to these subjects the prediction equations for maximal oxygen uptake, body potassium and oxygen uptake during submaximal exercise which were obtained for the factory workers, are summarized in table 7. This has been prepared by use of the combined regressions for male and female subjects listed in the text: the results and conclusions with the regression relationships for the male subjects alone are, for practical purposes, identical. The table shows that the maximal oxygen uptake of these athletes may be predicted with reasonable accuracy from the body potassium and cardiac frequency at an oxygen uptake of 1.5l min^{-1}, equation (4); by contrast, the aerobic capacity is possibly under-estimated when the thigh muscle width and the forced vital capacity are substituted for the body potassium, equation (5), despite the forced vital capacity being 20 % above the average for men of this age and size. At the same time the observed body potassium is higher than is predicted on the basis of anthropometric measurements, equation (1), whilst the oxygen uptake during cycling at 900 kp m min^{-1} (147 W) is somewhat lower than predicted for less athletic subjects of similar weight, equation (2); these findings may reflect an association between athletic prowess and both the composition of the muscles and the economy of movement during exercise.

Discussion

The direct determination of the capacity for exercise is an accepted procedure for athletes, students of physical education, mature adolescents and others who habitually take much exercise. The majority of the male subjects in the present study fall into the latter category and their results are typical. The results in the athletes are a little lower than have been reported for top performers in their respective events by Saltin & Åstrand (1967). However, in the latter study the range of the maximal oxygen uptakes was relatively wide, so the present results are probably not unrepresentative. The female subjects were unusual in that, whilst they turned out to be highly motivated, the majority took very little exercise; that they were able to achieve in a single session an aerobic capacity which is technically acceptable is therefore of great interest. In these subjects, on account of their sedentary habits, the oxygen uptakes are lower than in most reported series; they are similar to those of Swedish housewives reported by Åstrand (1960).

The aerobic capacity during cycling is mainly that of the thighs and calves (Houtz & Fischer 1959), with smaller contributions from the back and the arms. The performance of these muscles reflects the effectiveness of their constituent fibres, their overall size and the ability of the lungs and circulation to supply them with oxygen. The customary extents to which the muscles are used affect all of these aspects of function. The effects of activity constitute physical training; these include hypertrophy of muscle fibres and an increase in their content of myoglobin, expansion of the capillary network in the muscles, an increase in the total amount of haemoglobin in the blood, enlargement and hypertrophy of the ventricles of the heart leading to an increase in their stroke output, improved re-distribution of the blood flow during exercise from non-essential regions of the body to the muscles and the skin and finally improved function of the lungs. Of these attributes in subjects of comparable age, the one which is most directly related to the capacity for exercise is the maximal stroke output of the heart. This is the main determinant of the maximal cardiac output. By contrast, the maximal cardiac frequency, when standardized for the age of the subject, shows relatively little variability though it is rather less in subjects with large than with small hearts, equation (3). These interrelationships permit the estimation of aerobic capacity by extrapolation to a maximal frequency of the regression of oxygen uptake on frequency during submaximal exercise. In the present study the estimated maximal oxygen uptake is, on average, 0.3 l less than that obtained by direct measurement; the difference is similar to that observed by Wyndham, Strydom, Maritz, Morrison, Peter & Potgieter (1959), but the variability in the present series is rather larger, especially for the male subjects (coefficient of variation for male and female factory workers respectively 10 and 6.5 %). The difference between the sexes is mainly due to the consumption of oxygen at the higher of the two levels of submaximal exercise being a smaller proportion of the maximal

oxygen uptake in the male than in the female subjects (mean values 63 and 76 % respectively). Some of the variability is also due to the relationship of oxygen uptake to cardiac frequency being curvilinear in some subjects at near to the breaking point of exercise (Wyndham *et al.* 1959; Davies 1968).

The extrapolation procedure for the prediction of the aerobic capacity may be improved by the substitution for the cardiac frequency of the cardiac stroke volume measured by a rebreathing procedure, such as that described by Farhi & Haab (1967); alternatively, the diastolic heart size obtained by radiography may be used; however, the use of the stroke volume is unlikely to eliminate variability which is due to the curvilinearity of the cardiac frequency relationship, since this appears to be a function of training (Davies *et al.* 1968). For subjects studied on a single occasion, the alternative use of an empirical, rather than a formal procedure for prediction, may be a means of reducing variation on this account; it has the further advantage of reducing the severity and duration of the exercise which the subject is required to undertake. In practice, for the cardiac frequency alone, the residual standard deviation as a percentage of the mean for the regression of aerobic capacity on the frequency at a constant oxygen uptake ($1.5\ l\ min^{-1}$) is rather greater than that for the extrapolation procedure, but better than that for the frequency at a fixed work level ($450\ kp\ m\ min^{-1}$); the difference is probably due to the oxygen uptake in the latter circumstances being a function of body weight, equation (2). The precision is increased by the inclusion in the prediction equations of additional variables.

As well as the performance of the heart, the aerobic capacity is related to the amount of muscle which can be brought into use. For any exercise this is likely to be related to the total amount of muscle in the body, though the proportion will vary for different types of exercise and the distribution is also important. The total body muscle may be estimated in order of increasing precision from measurements of lean body mass, of body cell mass and of the muscles themselves. The lean body mass may be estimated from body weight and measurements of skinfold thickness (Durnin & Rahaman 1967), but is in any case a relatively crude index as no separation is effected between the remaining constituents of the body. In the present study it has been replaced by the total body potassium, of which about half represents potassium in the muscles. This index has the advantage over the lean body mass of being independent of the weight of the skeleton and the volume of extracellular fluid. It is highly correlated with the maximal oxygen uptake and like it is correlated with the width of the thigh muscle measured directly. These relationships are in accordance with theory; they also provide evidence that the index of thigh muscle width adopted for the present study is probably a reliable guide to total body muscle. It may, therefore, be used for this purpose in future studies, at least until more information becomes available. In addition, the measurement provides a means for predicting the expected body potassium in young adults with a coefficient of variation of 5.2 %, equation (1). The usefulness of the relationship is not impaired by it having different coefficient terms for male

and female subjects and it is likely to have a number of applications in medicine. However, the sex difference is a reminder that at least one of the component variables is of different quality for the two sexes; thus the potassium content of the muscle tissue may differ for males and females, possibly on account of differences in the fat or water content of the muscle (cf. Flear & Florence 1963). Alternatively, the body weight may need adjustment for differences between the sexes in one of its components, for example the density of the bones. The present data cannot be used to distinguish between these alternatives; however, that the prediction equations for maximal oxygen uptake on body potassium and on limb or thigh muscle may be combined ignoring sex, suggests that the component measurements are also, to a large extent, interchangeable.

The contribution of forced vital capacity as an index of lung function to the prediction of aerobic capacity is also in accordance with theory and consistent with the early work of Peabody & Wentworth (1917). The index is also convenient; however, whether or not it is the most informative of the lung function indices for this purpose has still to be established. By contrast to the FVC, the absence of a significant contribution from the indices of static and explosive muscle force is unexpected in view of the contrary findings of Ismail *et al.* (1965). The difference may reflect the selection of subjects or the procedures adopted in the two studies. Alternatively, technical factors in the analysis may be responsible, since a significant (but in our view, misleading) relationship would have been obtained in the present study if the data for all the subjects had been combined instead of being analysed for males and females separately (cf. table 3).

The prediction equations for maximal oxygen uptake, equations (4) and (5), unlike that for body potassium, equation (1), are common to the two sexes. This carries the implication that the lower maximal oxygen uptakes in the females, compared with the males, is mainly the result of their having smaller organs including the lungs, heart and muscles; the performance of the organs in relation to their size is essentially similar. This observation is relevant for the interpretation of comparative studies of exercise performance in people of different genetic constitution or environment. There is a need to look first for differences in organ size and to standardize for these before assessing the finer details of performance. The indices of physiological anthropometry which, from the present study, appear to be appropriate include the width of the muscle of the thigh, the vital capacity and the cardiac frequency at a constant uptake of oxygen; this should be for male subjects $1.5\ l\ min^{-1}$ and for female subjects $1.0\ l\ min^{-1}$. The work of Sjöstrand and other Scandinavian physiologists suggests that the diastolic heart volume, the total body haemoglobin and transfer factor should also be taken into account (see, for example, Holmgren & Åstrand 1966). The validity of the present approach is confirmed by the results of more recent studies now in the course of analysis: these suggest that the prediction equations for aerobic capacity and body potassium, which are reported above, also obtain for other populations (Cotes & Davies 1969; Burkinshaw *et al.* in preparation). Meanwhile, the potential usefulness of

the approach may be seen by analogy with studies in related fields. Thus in relation to ethnic variations, differences have been observed in the regression of lung volume on standing height between groups of subjects in whom the regression of the transfer factor on height were apparently identical (Cotes & Malhotra 1965). This finding points to a difference between the two ethnic groups in either the anatomy of the lung capillaries or the proportion which are perfused with blood. Similarly, in relation to the effects of physical training, a temporary increase has been demonstrated in the physical working capacity relative to the total body haemoglobin (Holmgren, Mossfeldt, Sjöstrand & Strom 1960). This finding points to differences in half-times between components of the response to training. In both these instances the search for an empirical association between different aspects of performance has led to a better understanding of the underlying processes.

In the present study three observations have been made which fall into this category. The first is the similarity of the relationships for the two sexes which is discussed above. The second is the absence of an association between hand-grip strength and the width of the muscles in the arm or the breadth of the hand; this points to some other factor, for example skill, as being a major determinant of performance. The third is the finding that the body potassium in the three athletes is both higher than expected on the basis of studies in non-atheletes and, in conjunction with cardiac frequency, is a reliable guide to the aerobic capacity. When combined with the other findings this suggests that differences between individuals in body composition, as well as in the size of their muscles, may influence their maximal oxygen uptakes.

In conclusion, the inter-relationships between structural and functional attributes pertaining to exercise provide a means for application of scientific method within groups of subjects, as well as between groups that are dissimilar on account of their genetic constitution or exposure to contrasting environments. This application of physiological anthropometry is likely to lead to advances in human exercise physiology.

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PHYSIOLOGICAL EFFECTS OF REPEATED EXERCISE

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SUMMARY

1. The physiological responses to repeated continuous (progressive) exercise together with the relationship between continuous and discontinuous work tests on an upright bicycle ergometer have been studied in healthy male subjects.

2. The results show a marked decline in mean cardiac frequency (f_H) from 145 beats/min to 124 beats/min during the first four experiments at a given submaximal oxygen intake of 1·5 l/min and thereafter a smaller decline to reach 118 beats/min on the final (sixteenth) day of the investigation. This latter decrease was associated with a small but significant rise in maximum oxygen intake ($\dot{V}O_2$ max) which occurred from Day 8 onwards. Pulmonary minute ventilation at an oxygen intake of 1·5 l/min ($\dot{V}_{E\,1\cdot5}$) decreased by about 5 l/min immediately following the first occasion of submaximal work but thereafter remained unchanged. There was however no decrease in O_2 cost of exercise, as $\dot{V}O_2$ at a fixed rate of external work remained unchanged throughout the investigation.

3. At maximal effort there was no significant decrease in either $\dot{V}_E$max or $f_H$max, but the f_H at which the $\dot{V}O_2$ max was reached declined significantly from Day 8. Thus the asymptotic nature of the $f_H/\dot{V}O_2$ curve which was very pronounced on Day 1 virtually disappeared following the third visit to the laboratory. The accuracy of predicting $\dot{V}O_2$ max from f_H and $\dot{V}O_2$ increased noticeably from $-15{\cdot}4\pm8{\cdot}9\%$ on Day 1 to $-6{\cdot}5\pm10{\cdot}5\%$ on Day 7 and $-0{\cdot}9\pm3{\cdot}4\%$ on Day 15. The possible physiological basis and implications of the results are discussed.

4. No significant differences were found between continuous and discontinuous work. Thus in large scale population studies of work capacity or in the evaluation of training programmes in rehabilitation studies, a continuous test may be used but attention must be paid to familiarization procedures. At least three preliminary test periods are necessary before the results become reproducible and reliable.

The maximum aerobic power ($\dot{V}O_2$ max) is now widely accepted by exercise physiologists as a

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reference standard of cardiorespiratory fitness (Shepherd, Allen, Benade, Davies, Hedman, Merriman, Myhre, di Prampero & Simmons, 1968). However, in many situations it is either impossible or inadvisable to measure $\dot{V}O_2$ max directly and for this reason several standard procedures for the estimation of $\dot{V}O_2$ max based on a person's response to submaximal exercise have been developed. The most commonly used are those based on measurements of cardiac frequency and oxygen intake (Åstrand & Rhyming, 1954), ventilation (Sadoul, Durand & Aubertin, 1958), respiratory quotient (Issekutz, Birkhead & Rodahl, 1962), and finally levels of lactic acid in the blood (Williams, Bredell, Fleming, Morrison, Strydom, Ward & Wyndham, 1962). Unfortunately in many cases where only a single test is given the possible physiological contribution of habituation to, and learning of, the test exercise is ignored. Further, in practice, it is usual to give either a continuous exercise test in which the work load is progressively increased at set intervals of time (Wahlund, 1948; Balke, 1952), or a series of discontinuous work periods of increasing severity interspersed with suitable rest pauses to allow recovery from the previous exertion (Åstrand, 1960; Maritz, Morrison, Peter, Strydom & Wyndham, 1961). Another problem therefore is whether these two forms of exercise yield similar and comparable results.

Exercise tests are also being increasingly used in the medical field for diagnostic purposes (Jones, 1967) and in the evaluation of rehabilitation programmes and drugs. In the majority of investigations the patient is used as his own control and his response to exercise is studied before and after some standard procedure (e.g. injection of a drug). It is tacitly assumed that any changes observed are due to the treatment.

We have investigated the effects of a repeated continuous (progressive) submaximal and maximal test and the relationship between continuous and discontinuous exercise on a bicycle ergometer.

MATERIALS AND METHODS

The investigation was in two parts. In part I, five healthy male subjects (aged 17–23 years) were each studied on 16 days during a period of 3 weeks and performed submaximal and maximal exercise on alternate days. The subjects were volunteers and a condition of enrolment was that they had never taken part in an experimental investigation or pedalled a bicycle ergometer before. All measurements were made with the subjects resting, but not basal, and no observations were obtained earlier than 1 h after a meal.

On the first visit to the laboratory the subject was instructed in the use of the mouthpiece and how to ride the Müller bicycle ergometer and how long he would be required to work. During the preliminary period the necessary electrodes were fitted and the subject rested in a chair for 15 min, during which period the cardiac frequency (f_H) was monitored. The lightweight recording electrodes (Devices Sales Ltd) were applied to each side of the chest in the midclavicular line about $\frac{1}{2}$ in. below the lowermost insertion of the pectoralis major and a third (earthing) electrode over the xiphoid process. This electrode arrangement was found to reduce muscle noise to a minimum even during maximal exercise and thus a clear recording of f_H was always obtained. At zero time the subject was asked to mount the bicycle and to pedal for 30 min at a pedal frequency of 60 revolutions/min. The work load was increased in intensity at the end of each 6 min period, the aim of the test exercise being to span an f_H range of 110–170 beats/min. The first three work loads were identical for each subject (300 kpm/min,

600 kpm/min and 900 kpm/min) but the final two work loads were adjusted in the light of the subject's probable $\dot{V}O_2$ max as judged from his response to the first stages of the test. The fourth work load ranged from 960 to 1200 kpm/min and the last from 1020 to 1380 kpm/min. Once the individual pattern of increase in work load has been established on Day 1 it remained constant throughout the rest of the investigation.

For maximal work the resting procedure remained the same. Exercise commenced with a 4 min warm-up at a work load corresponding to approximately 60% of the subject's probable $\dot{V}O_2$ max (f_H approximately 150 beats/min), the work load was then increased to approximately 90% of his $\dot{V}O_2$ max for 2 min and thereafter increased every minute until exhaustion was reached. The aim was to collect four sets of data at and beyond the subject's $\dot{V}O_2$ max. The criterion of maximum performance was that $\dot{V}O_2$ showed no further rise with increasing work load, i.e. at least two $\dot{V}O_2$ values were required to agree within $\pm 5\%$.

Throughout the resting submaximal and maximal work periods cardiac frequency was recorded continuously after the method of Davies & Neilson (1965). Expired air was collected during the fourth to sixth min at each submaximal work load and the final 45 s of each minute at maximal effort. The subject was connected via a low resistance Otis-McKerrow valve (dead space <100 ml) and a short length of smooth $1\frac{1}{8}$ in. internal bore tubing, to a wide-necked vinyl plastic Douglas bag which was suspended to reduce its resistance to flow (Cotes, 1966). The total resistance to expired gas flow was $<1{\cdot}5$ cm H_2O at 200 l/min. The bag was emptied through a dry gas meter (Parkinson-Cowan Ltd.) and a sample taken into small (1·5 l capacity) plastic bags for subsequent analysis of O_2 and CO_2 content using a paramagnetic O_2 analyser (Servomex Control Ltd) and infra-red CO_2 analyser (Beckmans Ltd). The analysers were calibrated before and after every reading with Standard gases which had to be previously analysed by the Lloyd-Haldane chemical method. The details of accuracy and reliability which might be expected from the method have already been given (Davies & Shirling, 1967).

In part II of the investigation, eleven subjects (aged 17–42 years) were studied on four separate occasions during continuous and discontinuous exercise. The protocol for the continuous work and methods for measuring cardiac frequency and expired air volume and content were identical to those described for part I of the study. The five work loads for the discontinuous tests were the same for the continuous work except that rest pauses (approximately commensurate with the severity of effort) of 10, 15, 20 & 30 min were allowed between each exercise period.

RESULTS

Submaximal exercise

Oxygen consumption

The regression lines of oxygen intake ($\dot{V}O_2$) on work load (W) for each subject for the first and final submaximal experimental occasion are shown in Fig. 1. The mean $\dot{V}O_2$ data at a W of 900 kpm/min ($\dot{V}O_{2\ 900}$) are summarized in Table I. They are substantially in agreement with the data of Åstrand (1960) for pedalling the stationary ergometer. The calculated mechanical efficiencies of our subjects were very close to the normal 23% (range 20–25%). There is some slight evidence from our figures to suggest that there may have been a small improvement in mechanical efficiency in three of the subjects (Fig. 1). However, neither the individual

nor the group regression lines of $\dot{V}O_2$ on W are significantly different from Day 1 to Day 16 ($P>0{\cdot}5$) and thus the $\dot{V}O_2$ at given W remained unchanged throughout the experimental period.

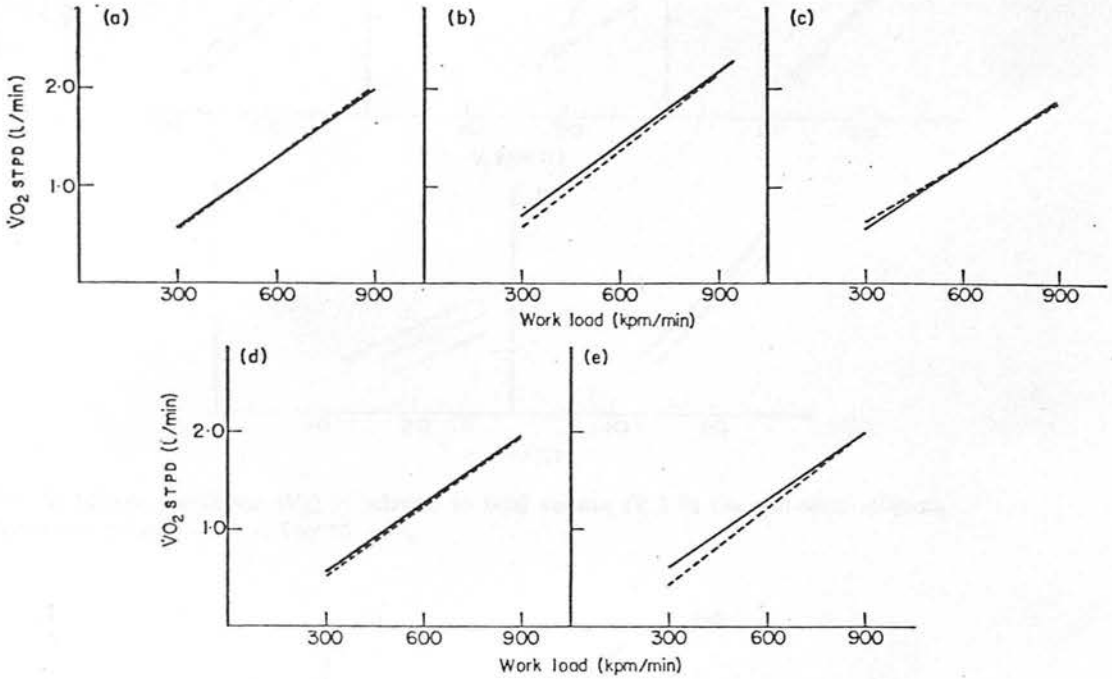

FIG. 1. Oxygen intake ($\dot{V}O_2$) in relation to work load in the individual subjects. Day 1 ——; Day 15 – – –.

TABLE 1. The effect of repetition on oxygen at 900 kpm/min ($\dot{V}O_{2\ 900}$), ventilation ($\dot{V}_{E\ 1{\cdot}5}$) and cardiac frequency ($f_{H\ 1{\cdot}5}$) at oxygen intake of 1·5 l/min. (mean data)

Day	1	3	5	7	9	11	13	15
$\dot{V}O_{2\ 900}$ (ml/min) STPD	1978	2021	2040	1990	2052	2022	1995	1976
$\dot{V}_{E\ 1{\cdot}5}$ (l/min) BTPS	40·46	35·79*	35·72	37·85	34·83	34·45	34·97	35·42
$f_{H\ 1{\cdot}5}$ (beats/min)	145	134**	131**	124**	124	125	117*	118

** $0{\cdot}01>P>0{\cdot}001$; * $0{\cdot}05>P>0{\cdot}01$

Ventilation

$\dot{V}_E/\dot{V}_T$ *relationship.* In Fig. 2 the expired ventilation ($\dot{V}_E$) is plotted against the tidal volume ($\dot{V}_T$) for the first, seventh and final submaximal experiments for each subject. A linear relationship was found in each experiment which could be expressed as $\dot{V}_E = m(\dot{V}_T - k)$ where m is the slope of the line and k the intercept on the $\dot{V}_T$ axis (Hey, Cunningham, Bolton, Jukes & Lloyd, 1966). Subject D showed a decrease in k over the experimental series, subject A showed an

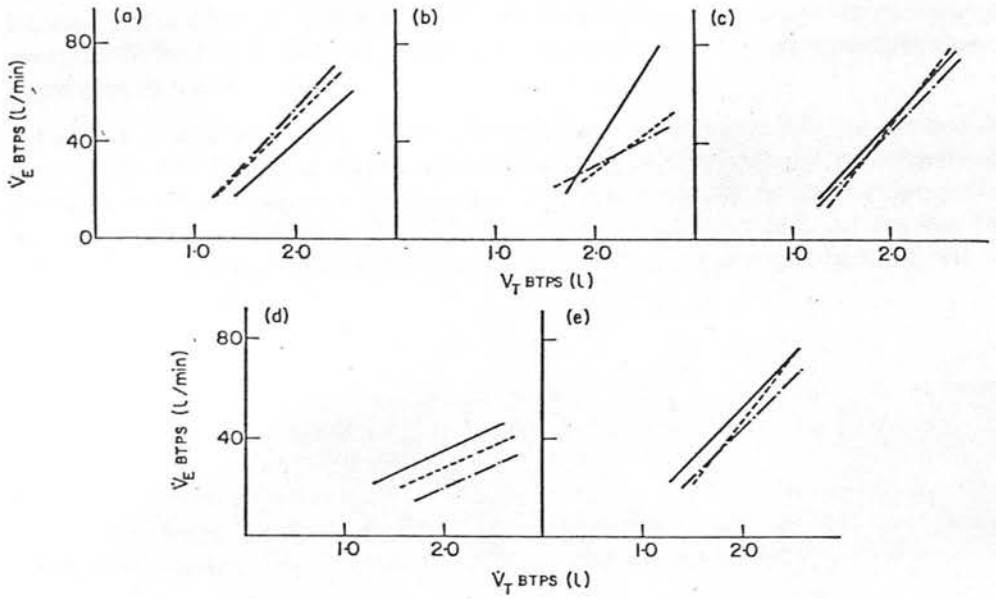

FIG. 2. Minute ventilation ($\dot{V}_E$) in relation to tidal volume ($\dot{V}_T$) in the individual subjects. Day 1 ——; Day 7 – · – · –; Day 15 – – –.

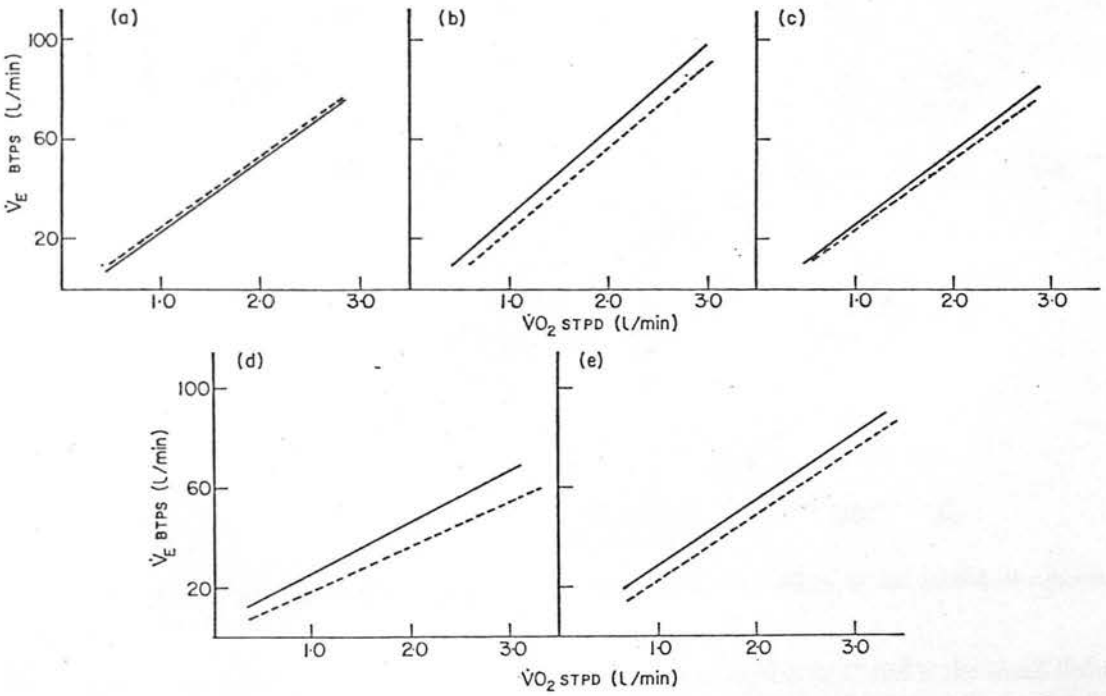

FIG. 3. Ventilation ($\dot{V}_E$) in relation to oxygen intake ($\dot{V}O_2$) in the individual subjects. Day 1 ——; Day 15 – – –.

increase and subjects C and E showed very little change, the parameter m remaining nearly constant. Subject B showed variability in both parameters. The grouped data show no significant overall trend.

Ventilatory equivalent. The individual regression lines of $\dot{V}_E$ on $\dot{V}O_2$ for the first three work loads in the first and final experiments are shown in Fig. 3 for five of the subjects. Subjects A and C show little change but the pooled data for the five subjects shows a significant decrease in $\dot{V}_E$ at a $\dot{V}O_2$ of 1·5 l/min ($\dot{V}_{E,\ 1·5}$). This decrease occurred between the first and second sub-maximal exercise experiments (Day 1 and Day 3) and thereafter the ventilatory equivalent remained unchanged.

Cardiac frequency

The cardiac frequency (f_H) data are summarized in Fig. 4 and Table I. The cardiac frequency at an oxygen intake of 1·5 l/min ($f_{H\ 1·5}$) decreased significantly ($P<0·001$) from a mean value of 145 beats/min on Day 1 to 124 beats/min on Day 7 and thereafter showed a smaller decline to reach 118 beats/min on Day 15 (Table I). From Day 1 to Day 7 the fall in $f_{H\ 1·5}$ was entirely due to the change in the intercept of the regression of the f_H on $\dot{V}O_2$, the slope of the line remaining constant (Fig. 4). From Day 7 onwards the individual changes in the slope and inter-

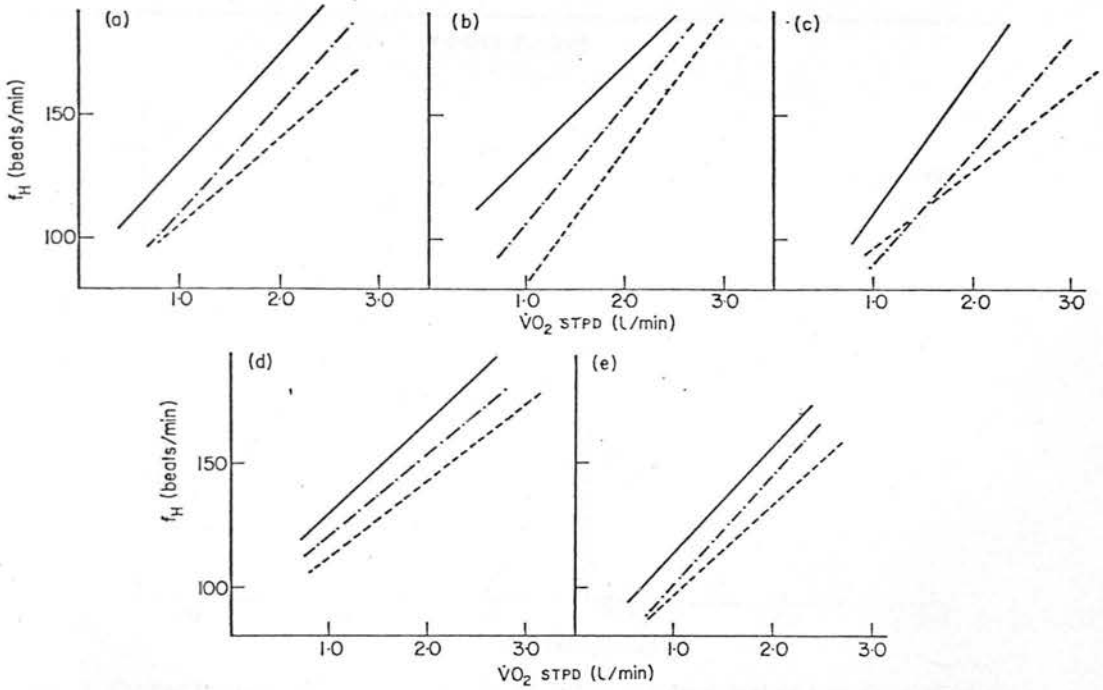

FIG. 4. Cardiac frequency (f_H) in relation to oxygen intake ($\dot{V}O_2$) in the individual subjects. Day 1 ——; Day 7 – · – · –; Day 15 – – –.

cept of the $f_H/\dot{V}O_2$ line were more variable (Fig. 4). In subjects A, C and E the small decrease in f_H was due to changes in slope and not intercept, whereas in subject B the reverse was true and subject D showed changes in both parameters.

Maximal exercise

The data from the maximal exercise on Days 2–16 are summarized in Table 2. Maximum oxygen intake ($\dot{V}O_2$ max) showed no significant change during the first three occasions of maximal work. However between Day 6 and Day 8 a small but significant rise in $\dot{V}O_2$ max occurred but thereafter it remained unchanged throughout the rest of the experimental period. The increase was of the order of 200 ml (2·6 ml/kg) which represents a 5·5% change in the measured $\dot{V}O_2$ max.

TABLE 2. The effect of repetition on maximum oxygen intake ($\dot{V}O_2$ max), cardiac frequency ($f_H$max), ventilation ($\dot{V}_E$ max), cardiac frequency at $\dot{V}O_2$ max (f_H at $\dot{V}O_2$ max) and ventilation at $\dot{V}O_2$ max ($\dot{V}_E$ at $\dot{V}O_2$ max)

Day	2	4	6	8	10	12	14	16
$\dot{V}O_2$ max (l/min) STPD	3·18	3·17	3·23	3·33*	3·42*	3·31	3·41	3·45
$\dot{V}_E$ max (l/min) BTPS	142·82	143·14	152·20	146·94	149·24	148·28	149·94	152·84
$\dot{V}_E$ at $\dot{V}O_2$ max (l/min) BTPS	123·04	129·81	120·47	121·15	121·30	113·64	112·85	111·45
f_H max (beats/min)	197	200	197	196	197	196	195	193
f_H at $\dot{V}O_2$ max (beats/min)	196	198	196	193*	193	189*	190	189

* $0{\cdot}05 > P > 0{\cdot}01$

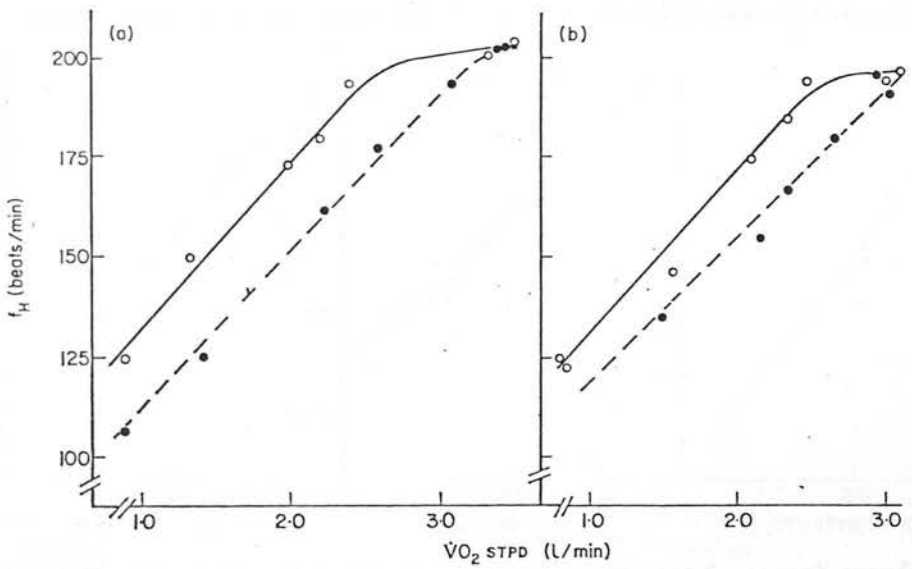

FIG. 5. Cardiac frequency (f_H) and oxygen intake ($\dot{V}O_2$) at submaximal and maximal effort. Days 1 and 2 ——; Days 4 and 5 – – –.

The mean maximum ventilation ($\dot{V}_E$ max) was 148·18 l/min (range 142·82 to 152·84 l/min) and showed no significant changes throughout the investigation (Table 2). However the $\dot{V}_E$ at which the $\dot{V}O_2$ max was reached did show a decline of 12 l/min over the 16 day period

(Table 2). This was also true of the corresponding cardiac frequency values; whereas the $f_H$max remained unchanged (mean 196 beats/min; range 193–200 beats/min) the f_H at the $\dot{V}O_2$ max decreased markedly from Day 8 onwards, reaching 189 beats/min on the final day of measurement. This significant decline in f_H($P<0{\cdot}01$) was thus associated with the small rise in $\dot{V}O_2$ max previously noted. These changes in f_H at $\dot{V}O_2$ max together with the decline in f_H at given $\dot{V}O_2$ resulted in a dramatic change of the $f_H/\dot{V}O_2$ relationship over the 16 day period of repeated exercise. The asymptotic nature of $f_H/\dot{V}O_2$ curve which was only pronounced on Day 1 began to disappear and was completely absent by the end of the investigation (Fig. 5). The accuracy of predicting $\dot{V}O_2$ max from submaximal values of f_H and $\dot{V}O_2$ increased from $-15\pm9\%$ on Day 1 to $-1\%\pm3\%$ on Day 15 (Table 3).

TABLE 3. Indirect estimation of maximum aerobic power ($\dot{V}O_2$ max) (see text)

Day	2	4	6	8	10	12	14	16
(i) $\dot{V}O_2$ max l/min (observed)	3·18 ±0·17	3·17 ±0·27	3·23 ±0·20	3·33 ±0·26	3·42 ±0·17	3·31 ±0·15	3·41 ±0·15	3·45 ±0·19
Day	1	3	5	7	9	11	13	15
(iii) $\dot{V}O_2$ l/min (predicted)	2·68 ±0·23	3·01 ±0·16	3·16 ±0·05	3·07 ±0·13	3·32 ±0·28	3·27 ±0·31	3·52 ±0·42	3·43 ±0·19
(ii)-(i) as % of (i)	−15·4 ±8·9	−4·0 ±13·8	−2·4 ±6·6	−6·5 ±10·5	−2·7 ±12·3	−1·1 ±11·5	+2·4 ±15·1	−0·9 ±3·4

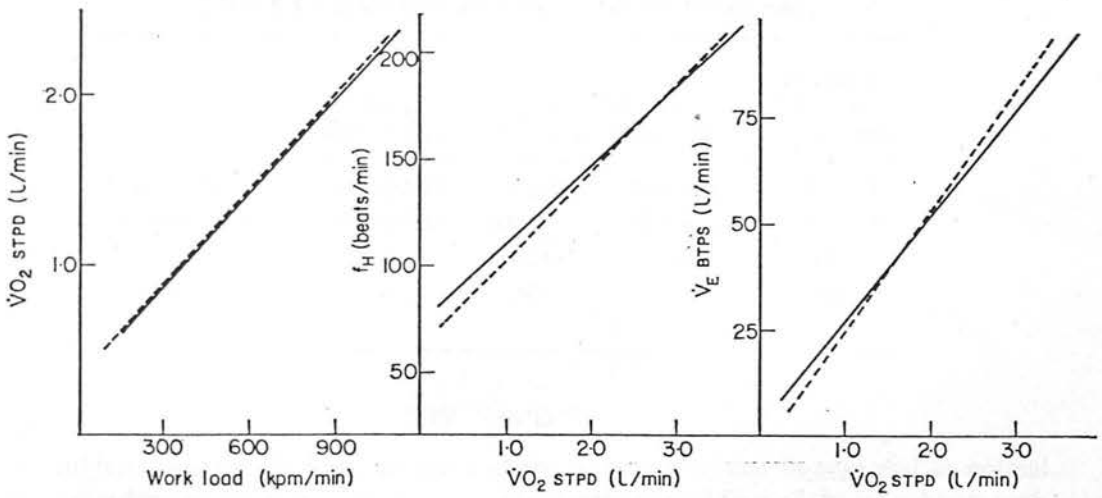

FIG. 6. Comparison between continuous (– – –) and discontinuous (——) work procedures. Oxygen intake ($\dot{V}O_2$) on work load; cardiac frequency (f_H) on $\dot{V}O_2$ ventilation ($\dot{V}_E$) on $\dot{V}O_2$.

The $\dot{V}_E/\dot{V}_T$ plots for all the subjects showed the expected linear relationship for values of $\dot{V}_T$ below 2·5 l/min. At higher values of $\dot{V}_T$ the relationship was curvilinear, tending to be more asymptotic at about $\dot{V}_T$ 3·5 l/min.

At $\dot{V}O_2$ max there was a tendency for $\dot{V}_T$ to decrease and respiratory frequency to increase

as exhaustion was approached. Mean $\dot{V}_T$ for all experiments was 3·18 l on the penultimate work load and 3·09 l on the last work load (mean of 45 readings).

Comparison between continuous and discontinuous submaximal work

The comparison between discontinuous and continuous (progressive) exercise is shown in Fig. 6 and Tables 4 and 5.

It is clear that provided the subjects are familiarized with and habituated to the work, there is no significant difference between the two forms of exercise.

TABLE 4. Comparison of continuous and discontinuous work. Intercept (a) and slope (b) of the regression of (1) $\dot{V}O_2$ on W (11)f_H on $\dot{V}O_2$ and (111) $\dot{V}_E$ on $\dot{V}O_2$

	(1)		(11)		(111)	
	(a)	(b)	(a)	(b)	(a)	(b)
Continuous	326·1	1·86	64·9	0·041	−2·78	0·028
Discontinuous	334·9	1·81	73·8	0·036	1·54	0·025
F_{1100}	1·39	0·33	1·44	1·50	0·18	0·76
Significance	NS	NS	NS	NS	NS	NS

TABLE 5. Comparison of continuous and discontinuous work

	$\dot{V}_{E1·5}$ (l/min) BTPS	$f_{H1·5}$ (beats/min)	$\dot{V}O_{2\ 900kpm/min}$ (ml/min) STPD	Predicted $\dot{V}O_2$ max. (ml/min) STPD
Continuous	38·61 ± 5·25	126 ± 11	2010 ± 120	3091 ± 475
Discontinuous	39·25 ± 6·16	128 ± 8	1973 ± 113	3110 ± 494
f_{10}	0·48	0·94	1·38	0·27
Significance	NS	NS	NS	NS

DISCUSSION

The six subjects forming the basis of part I of the investigation can be regarded as normal healthy but sedentary subjects. None was in athletic training and four of the six subjects took no regular exercise at all. Their lack of fitness is shown in the relatively low $\dot{V}O_2$ max values (Table 2) and the very high f_H reached during exercise of low to moderate intensity. The selection of subjects with a low working capacity was deliberate because in our experience it is precisely these people one normally encounters in population studies and in hospitals and laboratories where exercise tolerance tests are given.

The response of our subjects to repeated submaximal exercise may be divided into two

stages: (1) Days 1–7 during which changes in the physiological responses to standardized submaximal work occurred without an observable change in the $\dot{V}O_2$ max and (ii) Days 8–16 in which changes observed could be associated with an increased $\dot{V}O_2$ max.

Stage 1 (*Days* 1–7)

During the first three occasions of submaximal work, though the $\dot{V}O_2$ at a fixed work load and the VO_2 max remained constant, the circulatory system showed evidence of a profound change. The f_H at given $\dot{V}O_2$ of 1·5 l/min decreased from a mean value of 145 beats/min on Day 1 to 124 beats/min on Day 7. Ventilation at fixed oxygen consumption ($\dot{V}_{E\ 1·5}$) on the other hand, though it declined from 40 l/min to 36 l/min following the first occasion of submaximal work, remained fairly constant thereafter.

The fall in $f_{H\ 1·5}$ cannot be attributed to a decrease in the external work performed by our subjects due to a rise in mechanical efficiency or increase in overall work capacity since neither the $\dot{V}O_2$ at given work load nor the $\dot{V}O_2$ max changed during the 7 day period. Nor is habitation as conventionally defined—the change of f_H at a fixed $\dot{V}O_2$ due to decreased anxiety—a plausible explanation of our results since one would have expected to see the more pronounced changes in f_H at the lower work levels and for the effect to diminish with increasing severity of effort. The major changes in f_H 1·5 were due to a parallel displacement of the $f_H/\dot{V}O_2$ line (Fig. 2), intercept of the line being reduced but the slope remaining unchanged. One is therefore left with the conclusion that these changes in f_H must reflect some internal readjustments of an hitherto 'sedentary' circulation to demands of repeated and unaccustomed exercise.

The transport of oxygen to the working muscles is governed by the equation: $\dot{V}O_2 = Q \times AVD = f_H \times SV\ (Ca,o_2 - C\bar{v},o_2)$ where $\dot{V}O_2$ = oxygen intake; Q = cardiac output; AVD = arterio-venous difference; f_H = cardiac frequency; SV = stroke volume; $Ca,o_2 = O_2$ content of arterial blood and $C\bar{v},o_2 = O_2$ content of mixed venous blood. During submaximal work it is known that f_H and Q vary as linear functions of exercise intensity (Donald, Bishop & Wade, 1954) whereas the relationship of AVD and SV to $\dot{V}O_2$ is hyperbolic. Since our subjects showed a decrease in f_H on the first three occasions of submaximal exercise (with unchanged work loads), this suggests that either they were incapable of maintaining an adequate SV during the exercise or had a lower than normal AVD, which in turn implies that a larger than normal volume of blood was used for a given oxygen intake. Both these factors could arise from the same cause.

An abnormally low AVD usually arises in conditions of venous pooling particularly in the skin and splanchnic regions (Bevegard & Shepherd, 1967). This will directly affect venous return. Since an adequate venous return and filling time of the ventricles in diastole are the main determinants of SV an impairment of either of these factors could result in a smaller than normal stroke output and a higher than normal cardiac frequency.

The literature on the dynamic responses to training in young men is confused (Åstrand, 1956; Bevegard & Shepherd, 1967), and from our results we have no way of deciding whether an increase in AVD or SV occurred. However we feel that the decrease of $f_{H\ 1·5}$ together with the complete disappearance of the asymptotic nature of the $f_H/\dot{V}O_2$ curve (Fig. 4) is at least consistent with an overall theory based on blood redistribution. It is probable that during the first few occasions of repeated work, particularly in sedentary subjects unused to exercise, a redistribution of blood flow in favour of the working muscles takes place, thus allowing maximum use of the available cardiac output. Such an effect is probably mediated via the

autonomic system and represents an inherent readjustment of the body's circulation to the requirements of exercise.

Stage 2 (8–16 *days*)

During the second week of repeated exercise a more 'normal' training response was seen. The $f_{H\ 1\cdot5}$ showed a small but gradual decline of the order of 6 beats over the 8-day period. This was associated with an equally small but significant increase in $\dot{V}O_2$ max. It is also interesting to note that during this period, although the $f_H$max, and $\dot{V}_E$ max remained constant, the f_H and $\dot{V}_E$ at which the $\dot{V}O_2$ max occurred decreased markedly. This suggests that during a sustained period of repeated work the actual limiting factor to maximal work may change. The presence of an asymptotic $f_H/\dot{V}O_2$ curve during the first two occasions of maximal exercise indicates that a small amount of oxygen was utilized by the muscles without a concomitant rise in heart output. This could have occurred by a further unsaturation of mixed venous blood and a widening of the AVD as already indicated or by a redistribution of the available Q occurring during the course of the exercise. Since it would seem that the muscles are able to accept additional oxygen for a brief period of time after the circulation has reached its maximum capacity, it would be fair to assume the limitation to further effort was central rather than peripheral. This view was supported by the subjects' reactions to the first and second maximum test. During the final minute of the test they showed evidence of severe stress, were cyanosed and usually complained of overall and complete exhaustion.

In contrast during the second week of maximal testing when the f_H at the $\dot{V}O_2$ max had begun to decline and the asymptotic nature of the $f_H/\dot{V}O_2$ disappear, the overall picture of the limiting factor to maximal exercise began to change. Though the subjects found the work subjectively easier and were able to pedal at higher rates of work, this complaint was not of exhaustion but of localized pain in their legs, particularly in the quadriceps region.

Practical implications

The true nature of the physiological mechanism underlying the observed changes in healthy subjects performing repeated exercise await further investigation, but the practical implication of our results is clear. In situations such as large population studies of physical working capacity or in laboratory studies of training, where both the observers' and subjects' time are limited, a continuous exercise procedure may be used. However attention must be paid to familiarization procedures. The increase of predicted $\dot{V}O_2$ max without a concomitant change in observed $\dot{V}O_2$ max during the initial stages of the study suggest that at least three preliminary test periods should be given to ensure reliable and reproducible results.

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4

An analysis of O_2 debt contracted in submaximal exercise

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DI PRAMPERO, P. E., C. T. M. DAVIES, P. CERRETELLI, AND R. MARGARIA. *An analysis of O_2 debt contracted in submaximal exercise.* J. Appl. Physiol. 29(5): 547–551. 1970.—The kinetics of O_2 uptake ($\dot{V}O_2$) in man performing a submaximal exercise, in the transition from light to heavier work appears to be faster ($t_{1/2}$ = 17 s) than in the transition from rest to work ($t_{1/2}$ = 30 s). The difference in $\dot{V}O_2$ at the onset of exercise, i.e., the gross O_2 debt, is reduced correspondingly. This reduction presumably does not take place at the expense of the net O_2 debt; this, being the expression of the amount of split phosphagen present in the muscle at steady state, should be related only to the work intensity, independent of the previous history of muscle activity. It is presumably due *a*) to the greater utilization of the O_2 stores of the body when the exercise starts from rest and *b*) to a certain amount of LA formation from glycogen. This has been found to take place to a limited extent at the onset of work, but not in the transition from light to heavier exercise. In the transition from mild to heavier work only the gross alactic O_2 debt is then affected. The velocity of the O_2 uptake in this condition would tend to approach the velocity of the contraction of the net alactic O_2 debt.

O_2 debt, gross and net

IT HAS BEEN FOUND in man that, in submaximal exercise not involving lactic acid production, the O_2 consumption during the transition from rest to work and during the immediate recovery period changes as an exponential function of time, the $t_{1/2}$ of the processes being of the same order of magnitude ~30 second (s) (1, 2, 7, 8, 12, 13). Similarly in the isolated dog gastrocnemius the O_2 uptake at the onset of exercise has been shown to be an exponential function, which however has a $t_{1/2}$ of only about 16 s (14); the decrease of the O_2 uptake process in the recovery phase is also faster than in man ($t_{1/2}$ = 20 s).

A slow fall in recovery $\dot{V}O_2$ implies a correspondingly larger O_2 debt. The difference between the O_2 debt found in man and in the isolated muscle has been attributed to the fact that the O_2 debt, as measured in man from the respiratory gas exchange is the gross O_2 debt, i.e., it includes a certain amount of O_2 drawn from the O_2 stores of the body (12). A distinction has therefore been made between gross and net O_2 debt; this last only is indicative of the energy drawn from sources other than oxidations and is given substantially, in submaximal work, by the amount of split phosphagen (ATP + CP) in the muscles (4, 10). A correction for the O_2 drawn from the stores was therefore brought on the gross debt to obtain the net debt, and a reduced $t_{1/2}$ value of 22 s was thus obtained (10).

Another factor that may contribute to the gross O_2 debt in man is the formation of lactic acid (LA) from glycogen, a process that may take place to a limited extent in the first 2–3 min at the onset of work even in submaximal exercise (15, 16).

It is possible that in the transition from rest to exercise the adjustments of the higher O_2 uptake in man are slower than in the transition from the steady state of an aerobic exercise to a more strenuous one, because in the last case such adjustments are at work though at a lesser extent; this would possibly also imply a change of the rate of glycolysis in the two conditions.

The purpose of this paper was to study comparatively the rate of O_2 uptake at the onset of an exercise of relatively high intensity reached when starting from rest or from a milder exercise. The LA production in the two cases was also investigated.

EXPERIMENTAL PROCEDURE AND METHODS

The experiments were performed on five subjects whose physical characteristics are given in Table 1.

The following experimental procedures were used. *a*) In the first set of experiments the subject was asked to step up and down a bench 23 cm high, 20 times/min; after 5 min, when a steady state was reached, he shifted to a higher bench (46 cm), maintaining the same stepping frequency. After 5 additional min at the higher work load the subject shifted again to the lower step, for 5 more min. *b*) As a control, the subject performed for 5 min only the exercise of low or high intensities starting from and ending with rest. *c*) In further experiments the subject reached the steady state pedaling on a bicycle at 60 rev/min, at a work load of 502 kg-m/min; the frequency of pedaling was then suddenly increased to 100/min, and the work load to 713 kg-m/min; after 5 min the frequency of pedaling was reduced to 85 rev/min, and the work load increased to 972 kg-m/min.

In all sets of experiments the O_2 uptake was determined during the transition: *a*) from rest to each work level, *b*) from a mild to a strenuous exercise, *c*) from each work level to rest, and *d*) from a strenuous to a milder exercise.

The gas exchange in the unsteady state was determined by recording the inspired air volume by means of a spirom-

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cter and by sampling in rapid succession, every 3–5 s, the expired air in pipettes from a mixing chamber; the composition of this was determined by means of a Scholander apparatus. A correction for the time lag from the mouth to the mixing chamber was introduced.

The O_2 uptake at steady state was measured by collecting in a spirometer the expired air in the last 2 min of exercise.

In some experiments the gas exchange of the subject was continuously followed, on a breath-by-breath basis, by means of a recording pneumotachograph and an Atlas M 3 mass spectrometer (sampling line, 2 m; time lag, 0.8–1.0 s; 90% of the response, ~40 ms). From the tracings of P_{O_2}, P_{CO_2} and the expired airflow, the O_2 uptake of the single breath was calculated.

The O_2 consumption and the lactic acid contribution to metabolism at the onset of exercise was studied on subjects walking on a treadmill for 3 min at +10% incline at different speeds (3.2–6.5 km/hr) resulting in an oxygen consumption of 40–90% of $\dot{V}_{O_2\,max}$. In some experiments the subjects started the exercise from rest, in others from a state of mild aerobic work ($\dot{V}_{O_2} \sim 20$ ml/kg·min). Blood was then withdrawn from the cubital vein at the 2nd and 4th min of recovery when presumably the LA was uniformly distributed in the body fluids (3). The lactic acid determination was made by means of an enzymatic method (5): the maximal value attained after the exercise was used to calculate the total amount of lactic acid produced per kilogram of body weight, assuming that this was ¾ the amount of LA per liter of blood (11).

RESULTS

The average total O_2 consumption at each work level is given in Table 2 as milliliter per kilogram·minute and liter per minute, and as a percent of the maximal O_2 uptake of the subjects.

In Fig. 1 the O_2 uptake has been plotted as a function of time for three subjects during the step exercise, in the transition from rest to each work level, in the transition from mild to heavier work, and in the transition from each work level to rest, or from heavy to milder work.

All data concerning the transition from rest to work or vice versa as given by the percentage of the difference between the initial and the final $\dot{V}_{O_2}$ have been summarized in Fig. 2, *A* and *B*, and the data obtained in the transition from mild to heavier work or vice versa in Fig. 3, *A* and *B*. In all cases the behavior of O_2 uptake can be described approximately by an exponential function, no difference being appreciable among the different types or intensities of work.

In both kinds of exercise, stepping and cycling, the kinetics of O_2 uptake in the transition from rest to work is approximately the same ($t_{1/2}$ = 27–30 s) independent of the work level, in accordance with previous findings (1, 2, 6–8, 12, 13).

In the transition from mild to heavier exercise the O_2 uptake increases appreciably faster, being $t_{1/2}$ = 17 s. In contrast, during the transition from heavy to milder work, the kinetics of O_2 uptake appears to be slower ($t_{1/2}$ = 40–45 s) than when shifting from work to rest ($t_{1/2}$ = 30 s).

LA production. The increase of blood lactic acid (LA), after 3 min of uphill walking, when a condition of steady state in O_2 uptake was reached, has been plotted in Fig. 4 against the net O_2 uptake at steady state. The LA is also expressed in milliliter of O_2 per kilogram of body weight

TABLE 1. *Physical characteristics of subjects*

Subj	Age, yr	Ht, cm	Wt, kg	$\dot{V}_{O_2\,max}$, ml/kg·min
FC	28	179	63	44
PC	33	179	79	39
JC	30	160	58	
PP	28	177	65	44
FV	21	180	78	49

TABLE 2. *Average total O_2 consumption at each work level*

Type of Exercise	O_2 Uptake		% of $\dot{V}_{O_2\,max}$ Range
	ml/kg·min	L/min	
Step I (23 cm, 20/min)	18.5		38–47
Step II (46 cm, 20/min)	32.5		66–83
Bicycle I (502 kg-m/min)		1.120	29–40
Bicycle II (713 kg-m/min)		1.590	42–57
Bicycle III (972 kg-m/min)		2.170	52–78

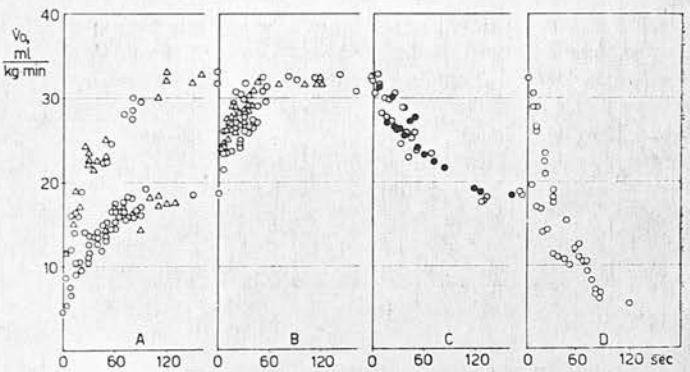

FIG. 1. Total O_2 uptake (ml/kg·min) as a function of time during the step exercise. *A:* transition from rest to either work level (23 cm, 20 times/min and 46 cm, 20 times/min, respectively); *B:* transition from milder to heavier work load; *C:* transition from higher to milder work; and *D:* transition from either work level to rest. Subjects: *PP*, ○; *FC*, △; *PC*, ●.

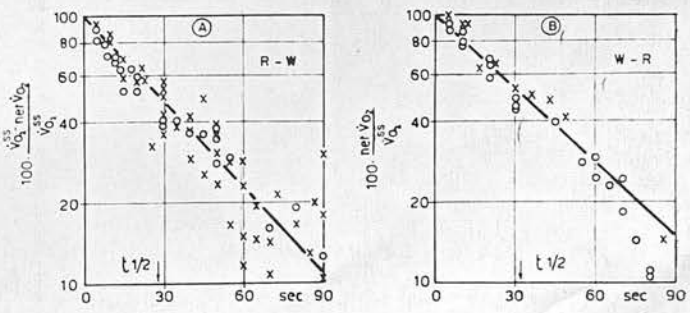

FIG. 2. Net O_2 uptake (above resting value) plotted as a function of time on semilog scale. *A:* rest to work (R − W); *B:* work to rest (W − R). O_2 uptake has been given as percentage of the difference between the two steady-state ($\dot{V}_{O_2\,ss}$) conditions, during the step exercise: ×, 23 cm, 20 times/min; ○, 46 cm, 20 times/min. Subjects as from Fig. 1.

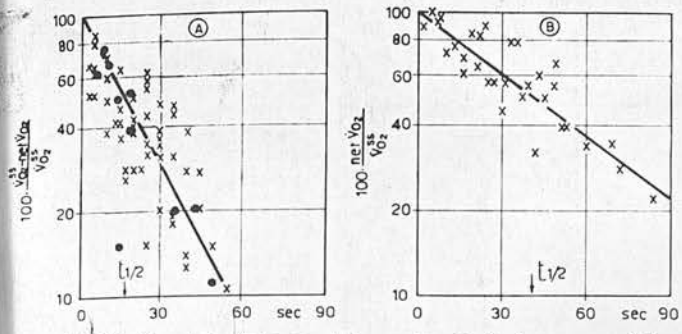

FIG. 3. Net O_2 uptake (above milder work value), in percent of the difference between the two steady-state ($\dot{V}o_{2\,ss}$) conditions, as a function of time, plotted on a semilog scale from *A:* milder to higher work load, and *B:* higher to milder work load. ×, Step, 23 to 46 cm and vice versa; ●, bicycle, 502 to 713 kg-m/min and 713 to 972 kg-m min. All subjects as from Table 1.

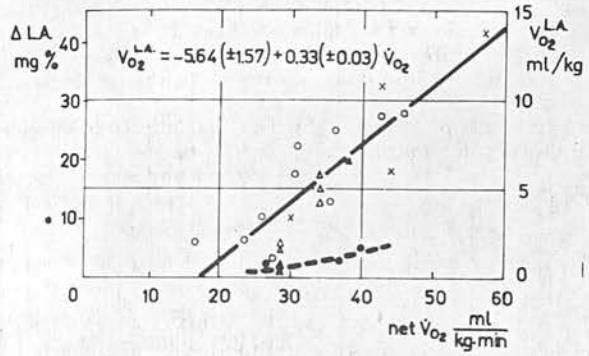

FIG. 4. Lactic acid concentration in blood (mg/100 ml) after 3 min f exercise as a function of net O_2 uptake at steady state. ○, Treadmill; ., step exercise; ×, data from Saiki et al. (15); ● refer to the experi- ents in which work load indicated on *abscissa* was reached starting ot from rest but from state of a mild aerobic exercise ($\dot{V}o_2$ = 20 l/kg·min).

$\dot{V}o_{2\,LA}$), on the assumption that 1 g of LA formed from ycogen is equivalent to 44 ml oxygen (11).

This function for the experiments concerning the transi- on from rest to work appears to be approximately a raight line, having the following formula, as calculated / the least-squares method:

$$\dot{V}o_{2\,LA} = -5.64\ (\pm 1.57) + 0.327\ (\pm 0.031)\dot{V}o_2 \qquad (1)$$

1 the experiments of transition from mild to heavier work, e lactic acid formed does not seem to increase significantly ıring the O_2 debt contraction phase (black dots of Fig.

CUSSION

The higher speed of the O_2 uptake process in the tran- on from mild to heavier work as compared with the nsition from rest to work implies a smaller alactic O_2 ɔt contraction. As mentioned earlier the "net" alactic debt is related only to the intensity of the exercise, matter whether this has been reached gradually or denly. Then in the transition from mild to heavier rk the smaller O_2 debt must be due to a lesser mobiliza- ı of O_2 from the body stores. Presumably the O_2 stores, ng reduced as a result of the mild work as compared to . are not depleted further to any significant extent in the transition to a heavier exercise. This hypothesis is supported by the fact that higher kinetics, of the same order of magnitude, have been found also in the isolated dog gastrocnemius, where the problem of the depletion of the body stores for O_2 is ruled out (4, 14).

It has been shown in a previous work (4, 10) that the gross alactic oxygen debt as measured in man ($\dot{V}o_{2\,g}$) is a linear function of the net oxygen consumption $\dot{V}o_2$ (12):

$$\dot{V}o_2 = k'\dot{V}o_{2\,g} \qquad (2)$$

where k', the speed constant of the payment of the gross alactic O_2 debt, amounts to 1.4 min^{-1}.

If the gross O_2 debt is corrected for the O_2 drawn from the body stores to obtain the net alactic debt ($\dot{V}o_{2\,n}$), the formulation above can then be written:

$$\dot{V}o_2 = k\dot{V}o_{2\,n} \qquad (3)$$

where k can be identified with the velocity constant of the process of phosphagen resynthesis: its value is k = 1.87, corresponding to a half-reaction time of 22 s (10). The corresponding value found in the isolated dog gastrocnemius is still lower ($t_{1/2}$ = 16.5 s) (14).

The value $t_{1/2}$ = 17.5 s found in the present experiments in the transition from mild to heavier work is similar to the value previously found in the isolated dog gastrocnemius. The discrepancy between these values and the value corrected for the O_2 drawn from the body stores seems to suggest that this correction is insufficient, or that other additional factors contribute to the gross alactic O_2 debt. In fact the gross oxygen debt ($\dot{V}o_2/k'$, as from *eq 2*), namely, all the oxygen missing at the onset work, besides being given by *a*) the net alactic oxygen debt, which can be expressed as from *equation 3* by $\dot{V}o_2/k$, and *b*) the oxygen that, coming from the oxygen stores of the body, escapes measurements at the respiratory level, ΔS, is also given for a small fraction by *c*) the lactic acid that is known to build up at the onset of a submaximal exercise during the oxygen debt contraction phase (15, 16). The gross oxygen debt may then be expressed by the sum of these three factors:

$$\frac{\dot{V}o_2}{k'} = \frac{\dot{V}o_2}{k} + \Delta S + \dot{V}o_{2\,LA} \qquad (4)$$

where $\dot{V}o_{2\,LA}$ is the LA expressed in O_2 equivalent. Then the lactic acid built in this period should be a linear function of $\dot{V}o_2$ provided that ΔS does not change appreciably in the transition from mild to heavier exercise. Thus from *equation 4:*

$$\dot{V}o_{2\,LA} = -\Delta S + \left(\frac{1}{k'} - \frac{1}{k}\right)\dot{V}o_2 \qquad (5)$$

or, giving to k' and k the experimentally found values 1.4 and 2.5, respectively, this last corresponding to $t_{1/2}$ = 17 s as found in the isolated muscle, as well as in the present experiments in the transition from mild to heavy work:

$$\dot{V}o_{2\,LA} = -\Delta S + 0.31\ \dot{V}o_2 \qquad (6)$$

The experimental data of lactic acid buildup in the transition from rest to exercise have been plotted as a function of the oxygen consumption at steady state in Fig. 4. The

line drawn through the points is described by the equation:

$$\dot{V}_{O_2\,LA} = -5.64\ (\pm 1.57) + 0.327\ (\pm 0.031)\dot{V}_{O_2}$$

The incline of the line, namely, the difference of the reciprocals of k' and k (see *eq 5*) appears to be substantially in agreement with the value as calculated (see *eq 6*).

The negative intercept of the line corresponds to the amount of O_2 drawn from the stores, and it amounts to 5.64 ml O_2/kg, or about 395 ± 110 for a 70-kg man. In *equation 3* the assumption is made that the depletion of the O_2 stores is constant, independent of the O_2 uptake of the exercise. This is acceptable within the range of O_2 uptake, above 20 ml/kg·min.

The depletion of the oxygen stores has been calculated in a previous work as amounting to 550 ml in supramaximal exercise (10). Considering that in the present experiments the work was submaximal, and the fraction of the O_2 stores due to myoglobin remained intact, a somewhat lower value appears to be reasonable.

In the transition from mild to heavier exercise the tissues should not undergo an appreciable anoxic state as the oxidative mechanism is already at work, and a steady state in oxygen consumption is reached faster; lactic acid production should then be nil. In effect the lactic acid produced in this condition is negligible (see Fig. 4).

According to Jöbsis and Stainsby (9), the LA production which occurs during the first minutes of stimulation of a mammalian skeletal muscle may be attributed to a temporary imbalance between pyruvate production and pyruvate removal by the tricarboxylic cycle. The inertia of the last process is less if the muscles start from a condition of moderate activity, and the two processes can then proceed at about the same rate.

On the basis of the above-mentioned considerations the time course of energetic processes taking place in the muscle in the first minutes of activity has been schematically given in Fig. 5.

Assuming that the contraction of both the gross and net O_2 debt processes is approximately an exponential function of time, and that this type of function is retained also when LA is produced, from the amount of the O_2 debt the speed constant can be calculated and the function drawn.

The three functions of Fig. 5 relate: *1*) to the contraction of the gross O_2 debt, *2*) to the contraction of the net alactic and lactic O_2 debt, and *3*) to the contraction of only the net alactic O_2 debt.

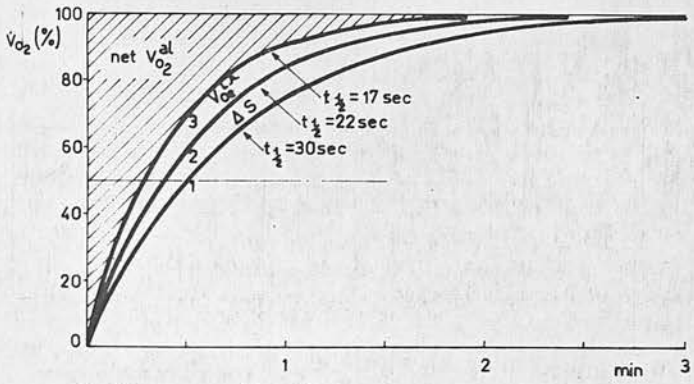

FIG. 5. Time course of net O_2 consumption at onset of aerobic work of medium intensity (50–70% of $\dot{V}_{O_2\,max}$) started from rest (schematic), and calculated from the assumption that the function is exponential, half-reaction times (or time constants) being as indicated. O_2 uptake at the lung level is given by curve 1 (k = 1.4 min^{-1} and $t_{1/2}$ = 30 s). As well as from the lungs, O_2 is also provided to muscles from body stores, and actual uptake of O_2 at the muscle level is given by curve 2 (k = 1.87 min^{-1} and $t_{1/2}$ = 22 s). By taking into account also lactic acid formed at onset of work (LA), time course of energy consumption as given by O_2 uptake and LA production is given by curve 3 (k = 2.5 min^{-1} and $t_{1/2}$ = 17 s). Hatched area indicates net alactic O_2 debt due to split phosphagen in muscle, whereas area between curves 3 and 2 is the fraction of O_2 debt due to LA formation and area between curves 2 and 1 is O_2 drawn from body stores.

The large area delineated by curve 1 and the coordinates indicates the gross O_2 debt. The area included between curves 1 and 2 is the expression of the depletion of the O_2 stores of the body. The area delineated by the curves 2 and 3 refers to the O_2 debt contraction due to LA formation. Finally, the area delineated by the curve 3 and the coordinates is the expression of the alactic net O_2 debt, as due to the splitting of phosphagen.

Slower kinetics of O_2 consumption in transition from strenuous to milder work. This, as compared with the transition from work to rest, seems statistically significant, and of the order of magnitude of 30%. Possibly more data on cardiac output and the O_2 content of mixed venous blood are needed to control the real existence and mechanism of this difference for which no satisfactory explanation is available at present.

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ESTIMATES OF ARTERIAL P_{CO_2} AND THEIR EFFECT ON THE CALCULATED VALUES OF CARDIAC OUTPUT AND DEAD SPACE ON EXERCISE

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SUMMARY

1. A study of exercise physiology in 117 children aged 6–16 is described including the measurement of cardiac output by the Indirect (CO_2) Fick Principle.

2. Computer produced graphs enabled various alternative values for arterial P_{CO_2} ($Pa,_{CO_2}$) to be inserted simultaneously into the Fick equation for cardiac output and the Bohr equation for dead space.

3. Dead space could be estimated with reasonable accuracy using end-tidal P_{CO_2}.

4. Cardiac output determination was unreliable at rest due to the small veno-arterial P_{CO_2} difference but it was very good on exercise when this difference is much larger.

5. The $Pa,_{CO_2}$ used for the calculation of cardiac output could be derived either from ear lobe P_{CO_2} or end-tidal P_{CO_2}. However, the best estimate of cardiac output was that using the $Pa,_{CO_2}$ implied by assuming a normal dead space.

Interest in the Indirect (CO_2) Fick method of measuring cardiac output has been revived by the studies of Campbell and his colleagues (Jones, Campbell, McHardy, Higgs & Clode, 1967; Higgs, Clode, McHardy, Jones & Campbell, 1967). The use of a rebreathing technique for estimating mixed venous P_{CO_2} ($P\bar{v},_{CO_2}$) renders this method simple, safe and acceptable even to children (Gadhoke & Jones, 1969), and it has been found valid compared with independent estimates of cardiac output (Higgs *et al.*, 1967; Ferguson, Faulkner, Julius & Conway, 1968).

McHardy, Jones & Campbell (1967) have pointed out that the transport of CO_2 from tissues to expired gas provided certain interrelationships between cardiac output and physiological dead space. This can be seen from the following versions of the equations of Fick (1870) and Bohr (1891):

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$$\dot{Q} = \frac{\dot{V}CO_2}{f(P\bar{v},CO_2 - Pa,CO_2)} \quad \text{(Fick)}$$

$$V_D = V_T \times \frac{(Pa,CO_2 - Pe,CO_2)}{Pa,CO_2} \quad \text{(Bohr)}$$

(where $\dot{Q}$ = cardiac output, $\dot{V}CO_2$ = CO_2 production, f = slope of CO_2 dissociation curve, $P\bar{v},CO_2$ = mixed venous PCO_2, Pa,CO_2 = arterial PCO_2, V_D = dead space, V_T = tidal volume, Pe,CO_2 = mixed expired PCO_2). All the values on the right hand side of these equations are easily determined without blood sampling, except for arterial PCO_2. However, if a value of arterial PCO_2 is substituted into the equations, simultaneous solutions for $\dot{Q}$ and V_D are obtained. In other words $\dot{Q}$, V_D and Pa,CO_2 are intimately linked together once the other variables are known. Use has been made of this fact to construct a digital computer program (Godfrey, 1970) to solve the simultaneous equations for any number of alternative estimates of Pa,CO_2.

In the present study we have explored the extent to which various alternative estimates of Pa,CO_2 affected the derived values for $\dot{Q}$ and V_D. This is of particular importance in order to interpret the response to exercise in subjects such as normal children in whom it is unjustifiable and often impractical to perform arterial catheterization.

MATERIALS AND METHODS

The study was carried out on a total of 117 children (fifty-seven boys and sixty girls) aged 6–16 years. They were all healthy volunteers from local schools. Each child was examined clinically before the study in order to exclude significant disease. Informed parental consent was obtained in writing for every child. Their physical details are reported elsewhere (Godfrey, Davies & Wozniak, unpublished observations).

Each child reported to the laboratory after a light breakfast or lunch and was studied at rest and $\frac{1}{3}$ and $\frac{2}{3}$ of its previously determined maximum working capacity. All measurements were taken when in the steady state, usually during the third to fifth min of exercise. Expired gas was collected for 1 min in a Tissot spirometer and immediately analysed for O_2 and CO_2. During the collection, or immediately afterwards in the more timid child, a sample of arterialized ear lobe blood was collected by the method of Godfrey, Wozniak, Courtenay Evans & Samuels (1970). This was followed by determination of the mixed venous PCO_2 ($P\bar{v},CO_2$), using essentially the method of Jones *et al.* (1967), except that the rebreathing bag was primed with CO_2 in O_2 mixtures from premixed gas cylinders which facilitated the selection of a suitable gas to achieve a plateau. The $P\bar{v},CO_2$ was taken to equal the plateau PCO_2 on the rebreathing record if it fulfilled the criteria of Ashton & McHardy (1963) and Jones *et al.* (1967). If not, the geometrical extrapolation procedure of Denison, Edwards, Jones & Pope (1969) was used. The next work level was begun immediately after completion of the rebreathing procedure.

Throughout the experiments O_2 was analysed with a paramagnetic analyser (Servomex O.A.150) and CO_2 with an infra-red analyser (URAS-4). These instruments were calibrated with four gas mixtures after every two or three patients. Ear lobe blood was analysed for PCO_2, PO_2 and pH with microelectrodes (Eschweiler) which were calibrated with three gas

mixtures and two standard buffers between each single sample. All calibrating gases were analysed in duplicate with a Lloyd-Haldane apparatus. The SD of the difference between duplicate estimates on the same sample of blood was 0·3 mmHg for P_{CO_2}, 0·9 mmHg for P_{O_2} and 0·005 units for pH. Correction for the difference between ear blood and arterial blood was made after the method of Godfrey *et al.* (1970).

Calculations

Analysis of mixed expired gas enabled the computer to calculate ventilation and gas exchange from the raw data for each subject at each work load. These calculations provided the variables $\dot{V}_{CO_2}$ and P_{e,CO_2} while $P_{\bar{v},CO_2}$ was measured from the rebreathing record and fed into the computer. The program then used these three constants together with stepwise increments of possible values for P_{a,CO_2} to produce a series of alternative solutions of the Fick and Bohr equations for the work load in question. In fact the relationship between $\dot{Q}$ and V_D is a smooth

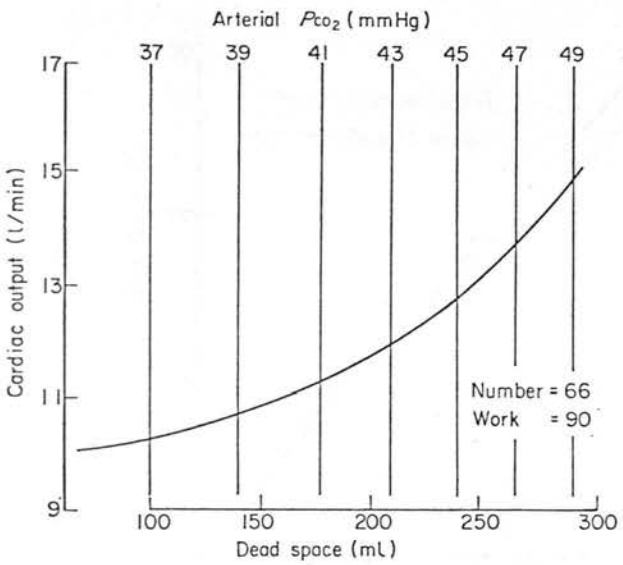

FIG. 1. Computer produced graph relating all possible alternative solutions of the Fick and Bohr equations for a given mixed venous P_{CO_2}, mixed expired P_{CO_2} and CO_2 production at one work load in one subject. Every point on the curved line represents the solution for cardiac output ($\dot{Q}$) and dead space (V_D) based on a particular value for arterial P_{CO_2} (P_{a,CO_2}). Some values of P_{a,CO_2} corresponding to points on the line are shown by the vertical lines.

curve, each point on the curve representing the solution for a different selected value of P_{a,CO_2}. A curve for one work load in a subject constructed by the computer is shown in Fig. 1. A similar curve was obtained for the other work loads performed by the subject and a separate set of curves was obtained for each subject. At each stage in the calculations, corrections were applied by the computer for the haemoglobin, arterial saturation, pH and base excess of the subject at the work load in question.

A graph, such as Fig. 1, produced for a subject at one work load makes it possible to insert any one of the three variables $\dot{Q}$, V_D and P_{a,CO_2} and then read off the other two. For example, if the likely value for V_D is known, the point on the curved line corresponding to this value of

V_D indicates the Pa,CO_2 which must have been present to satisfy the Bohr equation and the $\dot{Q}$ which is calculated by inserting this Pa,CO_2 into the Fick equation. A similar argument could be applied if Pa,CO_2 or $\dot{Q}$ were known instead of V_D.

In the present study, we used the graphs produced by the computer to read off two of the three variables $\dot{Q}$, V_D and Pa,CO_2 when the third was known as described above. For each work load in each subject five solutions were obtained from the graph using the following expected values as the known variable:

(a) Arterialized ear lobe blood PCO_2 fully corrected by the method of Godfrey *et al.* (1970).

(b) End-tidal PCO_2 uncorrected as an alternative to Pa,CO_2.

(c) End-tidal PCO_2 corrected by the formula of Jones, McHardy, Naimark & Campbell (1966) as an alternative to Pa,CO_2.

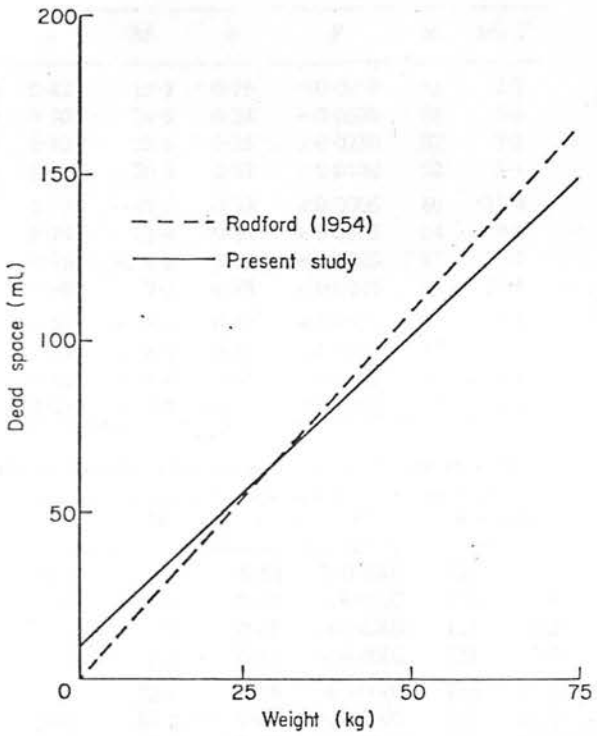

FIG. 2. The relationship between dead space (V_D) and weight (WT). The dotted line is that due to Radford (1954). The solid line is that for rest from the present study slightly modified to allow for tidal volume (V_T) according to the formula: V_D (ml) = 1·54 × WT (kg) + 0·049 × V_T (ml) + 2·0.

(d) $\dot{Q}$ appropriate to the oxygen consumption ($\dot{V}O_2$) of the work load in question from the relationship obtained by Bevegard, Holmgren & Jonsson (1960) for sitting bicycle exercise.

(e) V_D appropriate to body weight according to the relationship of Radford (1954) with our own modification for the effect of tidal volume (Fig. 2).

The five alternative solutions to the Fick and Bohr equations and the Pa,CO_2 which they implied for each work load in each subject were used to construct a series of regression equations in which the derived values were compared with the likely solutions. The likely solution for Pa,CO_2 was taken as the corrected ear lobe PCO_2 and used as the independent variable (X)

against which the four remaining estimates of Pa,CO_2 were each regressed as dependent variables (Y). The regression coefficient (B), the intercept on the Y axis (M), the correlation coefficient (r) and its significance (P) are all given for each of these equations in Tables 1 and 2.

TABLE 1. Regression equations for rest for arterial P_{CO_2}, dead space and cardiac output. Y is the dependent variable which is the parameter in the first column derived graphically from a normal dead space (V_D), a normal cardiac output ($\dot{Q}$), end-tidal P_{CO_2} (ET), corrected end-tidal P_{CO_2} after Jones *et al.* (1966)—(JET), or from ear lobe blood (Blood). X is the independent variable which is the assumed normal value for Pa,CO_2 based on ear lobe blood P_{CO_2} (Blood), the assumed normal value for dead space (V_D), or the assumed normal value for cardiac output based on oxygen consumption ($\dot{Q}$). The symbols are explained in the text

		Y	X	B	M	r	P	n	SE Y
	1	V_D	Blood	0·42	19·9	0·29	<0·0250	52	3·7
Arterial	2	$\dot{Q}$	Blood	0·30	24·9	0·24	<0·0500	51	3·3
P_{CO_2}	3	ET	Blood	0·32	22·1	0·28	<0·0250	52	3·1
	4	JET	Blood	0·40	20·5	0·33	<0·0100	52	3·1
	5	Blood	VD	1·57	−41·9	0·79	<0·0005	69	32·4
Dead	6	$\dot{Q}$	VD	0·74	21·4	0·55	<0·0005	84	29·3
space	7	ET	VD	0·96	− 4·1	0·78	<0·0005	87	20·2
	8	JET	VD	0·90	7·1	0·75	<0·0005	87	21·1
	9	Blood	Q	2·67	− 8·1	0·49	<0·0005	66	1·8
Cardiac	10	V_D	Q	1·99	− 4·7	0·50	<0·0005	84	1·4
output	11	ET	Q	1·80	− 4·4	0·67	<0·0005	84	0·8
	12	JET	Q	1·76	− 3·8	0·57	<0·0005	84	1·0

TABLE 2. Regression equations for exercise with the same nomenclature as Table 1

		Y	X	B	M	r	P	n	SE Y
	1	V_D	Blood	0·75	9·7	0·58	<0·0005	121	3·1
Arterial	2	$\dot{Q}$	Blood	0·59	16·6	0·44	<0·0005	116	3·6
P_{CO_2}	3	ET	Blood	0·86	3·7	0·61	<0·0005	121	3·2
	4	JET	Blood	0·54	15·3	0·48	<0·0005	121	2·9
	5	Blood	VD	0·56	22·1	0·34	<0·0005	153	54·9
Dead	6	$\dot{Q}$	VD	0·81	37·4	0·45	<0·0005	181	57·3
space	7	ET	VD	0·81	−20·7	0·66	<0·0005	193	31·7
	8	JET	VD	−0·42	92·8	−0·31	<0·0005	193	44·5
	9	Blood	Q	0·97	− 0·4	0·92	<0·0005	144	1·0
Cardiac	10	V_D	Q	0·99	− 0·2	0·94	<0·0005	182	0·9
output	11	ET	Q	0·97	− 0·6	0·94	<0·0005	182	0·9
	12	JET	Q	0·81	1·1	0·92	<0·0005	182	0·8

In addition the number of points (n) from which the data was constructed is given together with the standard error of the estimate of Y about the line (SE Y). The same procedure was used for dead space in which case the likely value for V_D used as the independent variable (X) was that based on body weight and tidal volume, and for cardiac output the likely value used (X) was that based on $\dot{V}_{O_2}$ as described above. The four regression equations for V_D and for $\dot{Q}$ are also given in Tables 1 and 2.

RESULTS

Using Table 1 (for rest) and Table 2 (for exercise) it was possible to make certain observations about the various alternative methods of calculating V_D, $\dot{Q}$ and Pa,CO_2.

Dead space

The highest correlation with the likely V_D based on weight was obtained by using the V_D calculated from ear blood P_{CO_2} ($r = 0{\cdot}79$) or from uncorrected end-tidal P_{CO_2} ($r = 0{\cdot}78$) at rest (Equations 5 and 7, Table 1). The simplest regression equation was that based on end-tidal P_{CO_2} ($B = 0{\cdot}96$, $M = -4{\cdot}1$) implying that the value of V_D calculated from uncorrected end-tidal P_{CO_2} at rest agreed closely with what is predicted from body size (Fig. 3). The error of

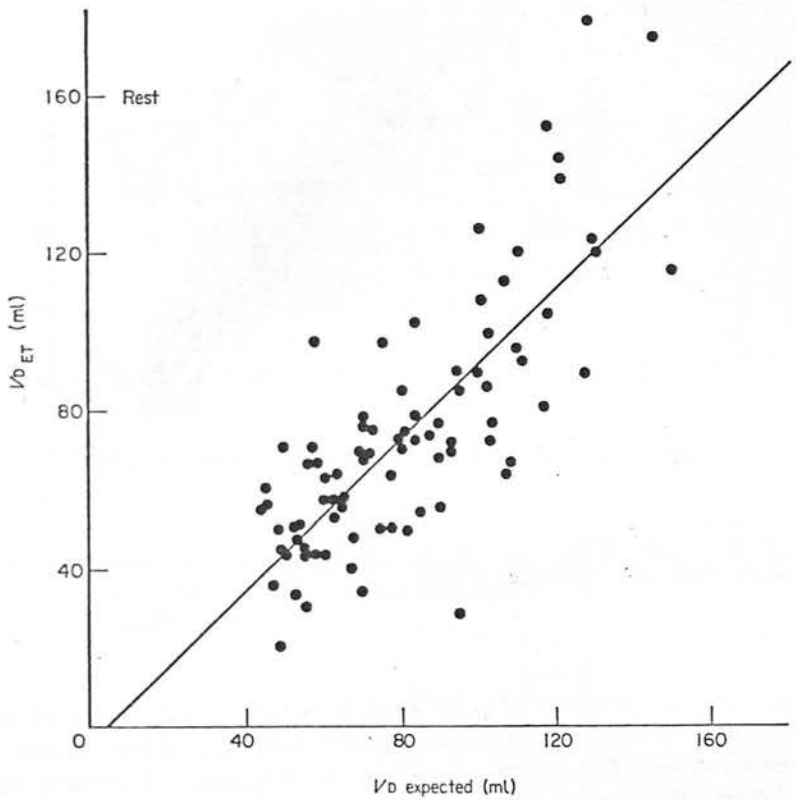

FIG. 3. The relationship between dead space calculated from end-tidal P_{CO_2} ($V_{D_{ET}}$) and that predicted from body weight and tidal volume ($V_{D_{expected}}$). These results are for rest.

all estimates of V_D (SE Y) was large. As far as exercise was concerned, the correlation coefficients were generally smaller than those at rest and the SE Y were larger, but again the estimate based on uncorrected end-tidal P_{CO_2} was the best (Equation 7, Table 2).

Cardiac output

Unlike V_D, the estimations of $\dot{Q}$ on exercise showed much greater agreement with the expected values than the estimations at rest (Equations 9–12, Tables 1 and 2). Indeed all the correlation coefficients were extremely high on exercise (0·92–0·94) with little to choose

between the four alternative equations and all the SE Y values were between 0·8 and 1·0 l/min. It is particularly important to note the excellent agreement between the $\dot{Q}$ calculated from ear lobe P_{CO_2} or from the P_{CO_2} implied by a normal V_D (Equations 9 and 10, Table 2) with the expected $\dot{Q}$ based on V_{O_2}. The latter relationship is shown in Fig. 4.

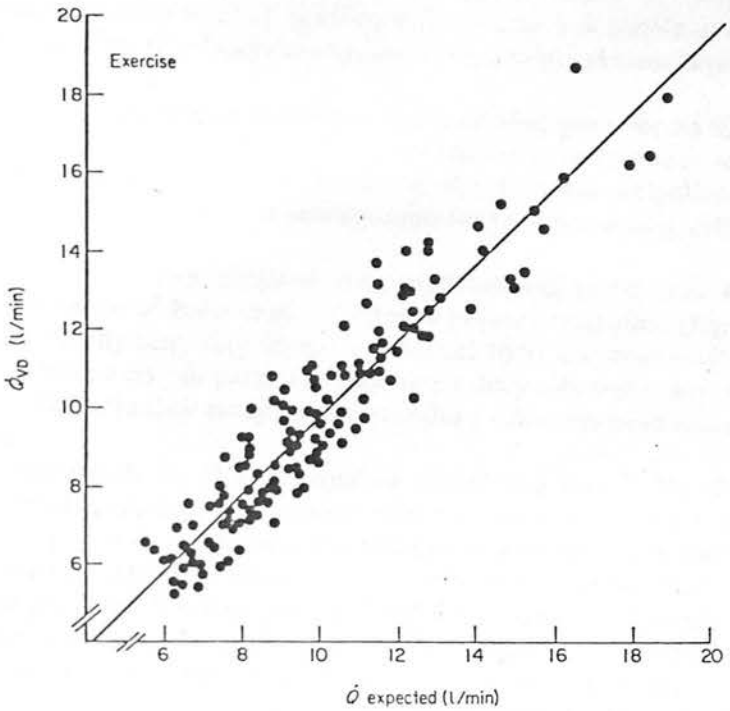

FIG. 4. The relationship between cardiac output calculated from the arterial P_{CO_2} implied by a normal dead space ($\dot{Q}_{VD}$) and the expected cardiac output predicted from oxygen consumption ($\dot{Q}_{expected}$). These results are for exercise.

Arterial P_{CO_2}

The comparison between various forms of arterial P_{CO_2} (Y) derived from end-tidal P_{CO_2} or implied by the assumption of a normal $\dot{Q}$ or V_D, with the measured (and corrected) ear lobe P_{CO_2} (X), was poor both at rest and on exercise (Equations 1–4, Tables 1 and 2). Like the equations for $\dot{Q}$ however, the exercise values were rather better than those at rest, the best equations being those based on an assumed normal V_D and on uncorrected end-tidal P_{CO_2} (Equations 1 and 3, Table 2), but even so the SE Y was of the order of 3 mmHg.

DISCUSSION

In order to investigate the various methods of deriving $\dot{Q}$ and V_D from the possible alternatives available in the CO_2 transport method of analysis a large number of calculations were necessary. The use of a digital computer to perform these calculations and to draw the graphs not only made the comparisons of this study possible, but also reduced mathematical errors to a minimum and vastly reduced the time spent on calculations.

The difference between the results at rest and on exercise for V_D and $\dot{Q}$ can be explained by the fact that the arterio–expired P_{CO_2} difference changes relatively little from rest to exercise, while the veno–arterial P_{CO_2} difference widens considerably. The former difference governs the calculation of V_D and being normally small (of the order of 10–15 mmHg), is hence susceptible to analytical errors both at rest and on exercise. The veno–arterial P_{CO_2} difference governing the calculation of $\dot{Q}$ enlarges from some 6–8 mmHg at rest to some 25–35mmHg on exercise. Thus $\dot{Q}$ is highly susceptible to any analytical error at rest but relatively insensitive on exercise.

The above reasoning also explains the somewhat poor results obtained for V_D based on ear blood P_{CO_2} since an error of 2 SD of the difference between true $Pa,_{CO_2}$ and ear blood P_{CO_2} (2·6 mmHg) as found by Godfrey *et al.* (1970) would significantly affect calculations of V_D at rest and on exercise. It also accounts for the poor resting values of $\dot{Q}$ derived from ear blood P_{CO_2}.

Our best results were obtained using end-tidal P_{CO_2} to calculate V_D but we found that the correction factor of Jones *et al.* (1966) gave poorer correlations (Equation 8, Tables 1 and 2). This is not really surprising since it was derived from studies in adults under conditions rather different from ours—in particular their respiratory rate was lower. Gadhoke & Jones (1969) used this factor in their study but their resulting values for dead space can be shown to be very variable.

Our best results for $\dot{Q}$ were based on a predicted normal V_D. This is helpful in children with normal lungs since V_D increases with weight (Radford, 1954; Tenney & Bartlett, 1967; Levinson, personal communication) and can be predicted with reasonable certainty (Fig. 2). Our results for $\dot{Q}$ will be discussed more fully later (Godfrey *et al.*, 1970), but the agreement with the predicted $\dot{Q}$ is encouraging. We did not apply the 'downstream correction' to $P\bar{v}_{CO_2}$ described by Jones *et al.* (1967) and Jones, Campbell, Edwards & Wilkoff (1969) but like Dennison *et al.* (1969) we found the uncorrected value to be more useful.

We conclude that the Indirect (CO_2) Fick method for $\dot{Q}$ gives reliable results on exercise, even in quite small children, virtually independent of the method of estimating $Pa,_{CO_2}$. The best method is to calculate $Pa,_{CO_2}$ from the Bohr equation using a normal V_D predicted from weight if the lungs are normal. Alternative results based on end-tidal or ear blood P_{CO_2} are acceptable. Calculations of $\dot{Q}$ at rest are most unreliable. Calculation of V_D from end-tidal or ear blood P_{CO_2} is moderately reliable.

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Computer quantitation of ST segment response to graded exercise in untrained and trained normal subjects

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Authors' synopsis *A computer technique has been employed to record continuously the amplitude of selected points on the ECG waveform during and after graded exercise in untrained and trained young subjects. There was a wide scatter of values, a proportion lying outside previously suggested criteria of normality. The general pattern was one of increasing ST segment depression and slope with increasing severity of exercise.*

The changes which occur in the electrocardiogram during and after muscular exercise are poorly documented except for certain indices conventionally used in the diagnosis of ischaemic heart disease. With increasing heart rate it is known that there is some shortening of the PR interval and of the QT interval. The duration of the QRS complex, however, remains almost unchanged. There is a voluminous literature on the response of the ST segment immediately after exercise. Distortion of the electrocardiographic signal by artefact has until recently prevented accurate quantitation of responses during exercise. Several investigators have reported, however, that the ST segment response during exercise gives information of greater diagnostic value in ischaemic heart disease (Rosenfeld and Master, 1964) and with the recent development of computer averaging techniques to reduce random noise in the electrocardiogram several studies are available quoting depressions of the ST segment averaged over a large number of complexes during exercise. These studies generally assume that the changes during and after exercise are relatively constant, and little is known about the time course of the ST segment changes during and after exercise. The purpose of the present study is to ascertain the time course of these changes during and after walking or bicycling exercise.

The two technical improvements which have led to the present interest in ECG recording during exercise are the development firstly of electrodes with a low mass which minimize noise due to electrode movement, and secondly of computer devices to remove noise from the ECG – for example, the CAT computers used by Doan, Peterson, Blackmon, and Bruce (1965) and by McHenry, Stowe, and Lancaster (1968). Such computer analysis allows study of the total response of the ST segment in terms of both depression and slope. It does, however, involve averaging a large number of complexes and does not therefore give a continuous record of the behaviour of the ST segment.

The method described in the present paper is an improvement upon this in two respects; firstly, it allows continuous monitoring of selected points on the ECG complex, and, secondly, the behaviour of an unlimited number of such points on the complex can be studied by repeated computer processing of the tape-recorded ECG signal. As a result, continuous recording of changes in the ECG can be produced in as complete detail as is required. We have used this method to study the response to two grades of treadmill exercise in healthy young subjects for comparison with cases of known ischaemic heart disease. The results show a surprising variability of the normal response, both in terms of ST segment elevation or depression and of changes in slope. They also show consistent

time-dependent changes during and after exercise which imply that different results could be obtained, depending on the time of sampling of the ECG, and which cast doubt on previous exercise ECG studies which sample the waveform at arbitrary times only. The degree of ST depression in many cases fell outside the limits previously adopted as normal criteria (Bellet, Eliakim, Deliyiannis, and La Van, 1962). This type of response has been recorded previously in subjects without evidence of ischaemic heart disease by Holmgren, Jonsson, Levander, Linderholm, Sjöstrand, and Ström (1959) and by Friesinger, Likar, Biern, and Mason (1965), and the suggestion has been made that it may occur in subjects in a poor state of physical training or during hyperventilation. The observations made on untrained subjects have therefore been repeated and extended in a second group of subjects in good physical training – that is, students of physical education.

Methods

1 Studies in untrained subjects

Studies were performed on 36 healthy subjects aged 20 to 24. Satisfactory recordings were obtained in 31 of these subjects and these have been used for analysis. Three of these subjects were female. They were studied in the post-absorptive state and at a room temperature of 16–19°C. Exercise was carried out on a motor driven treadmill. For each subject four 5-min periods were studied: (1) a control period of 5 min standing on the treadmill, (2) 5 min walking on the level at 4 mph, (3) 5 min walking at the same speed with a 10% tilt on the treadmill, following continuously on (2), and (4) a 5-min recovery period standing on the treadmill.

Light exercise increased the average heart rate from 92/min to 127/min and on severe exercise there was a further rise to 169/min. Heart rate fell immediately on stopping exercise and had returned to a stable level in 5 min.

The predicted maximum heart rate for individuals of this age is approximately 195/min (Åstrand, 1960; Davies, 1968). These were untrained subjects and no attempt was made to produce a maximal heart rate by exercise. The predicted average oxygen consumption corresponding to the two levels of exercise were 970 ml./min and 1,920 ml./min for a 70 kg subject 68 in. (172·7 cm) tall (Workman and Armstrong, 1964).

TABLE I *Subjects*

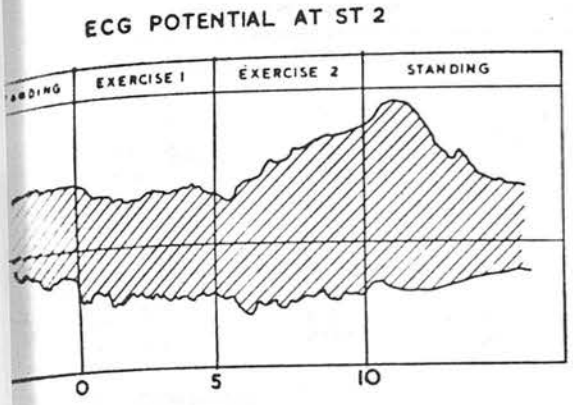
Height (*cm*)	*Weight* (*kg*)	*Age* (*yr*)	$\dot{V}o_2$ *max* (*l./min*)	$\dot{V}o_2$ *max* (*ml./kg*)
165·8	59·8	25·5	3·13	52·3
164·5	54·8	27·8	2·87	52·4
150·9	66·0	25·2	3·52	53·33
175·1	77·8	22·5	3·94	50·64
166·1	63·1	37·3	2·79	44·21
180·4	82·0	36·8	3·66	44·63
167·6	56·8	38·3	2·28	40·14
164·6	65·6	31·3	2·86	43·60
164·2	50·7	29·8	2·29	45·17
180·5	79·3	21·3	4·13	52·08
180·2	76·3	20·1	3·74	49·02
177·6	74·3	24·3	3·81	50·93
174·1	69·2	18·8	3·60	52·02
184·4	73·9	22·8	3·56	48·17
177·6	79·2	29·9	3·42	43·18
170·7	76·8	26·6	3·59	46·74
174·2	63·5	29·0	2·95	46·46
173·3	72·1	20·0	3·45	47·85
173·43 ±6·66	68·98 ±9·37	27·06 ±6·02	3·31 ±0·535	47·937 ±3·894

2 Studies in trained subjects

These were healthy male volunteers aged 20–30 years. They were students of physical education at the Carnegie College of Physical Education, Leeds. Their physical characteristics are given in Table I. The subjects were usually studied after a light breakfast or lunch. They were accustomed to the exercise procedure in order to avoid the problems of habituation to and learning of the given task (Davies, Tuxworth, and Young, 1968). For this purpose the subjects undertook two periods of 10-min warm-up exercise at 100 kpm/min separated and followed by 20 min of rest.

After this preliminary period, the subjects were asked to pedal an upright bicycle ergometer (Muller, Dortmund) at a frequency of 60 rev/min and at four different work loads of increasing severity. The work load was initially set at 300 kpm/min, then raised by 300 kpm/min after each 6 min period; the aim being to try to cover the full range of the subject's exercise capacity. After a further rest period the maximum aerobic power ($\dot{V}o_2$ max) of the subjects was assessed according to the method described by Davies (1968). The work intensity was increased every 3 min from a starting point which was determined in the light of the response of the subject to submaximum work. The maximum was judged to have been achieved, when upon subsequent analysis, an increase in work load caused no further rise in the consumption of oxygen – that is, at least two 'plateau' values agreed within ±5%.

The subjects inhaled through a low resistance Otis-McKerrow valve and the expired air was collected via a short length of smooth internal bore tubing into a wide bore vinyl plastic Douglas bag which was suspended to reduce its resistance. The dead space of the valve box was <100 ml. and the resistance to flow of the whole respiratory circuit <1 cm at a gas flow of 300 l./min. The Douglas bag was emptied through a dry gas meter (Parkinson and Cowan, Ltd). The $\dot{V}o_2$ during the submaximal exercise was determined over the 4–6th min and at maximum effort dur-



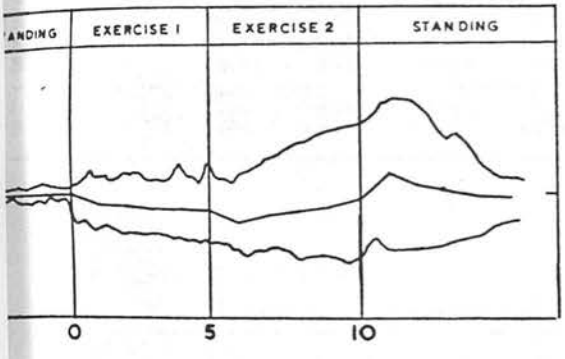
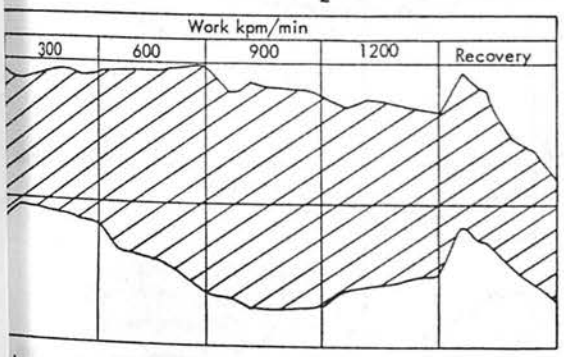


FIG. 1 Untrained subjects. a (above): *absolute values of potential at ST_2 with reference to PQ segment potential – range in 31 subjects.* b (below): *change in ST_2 potential on exercise, with reference to resting value – range and mean in 31 subjects.*



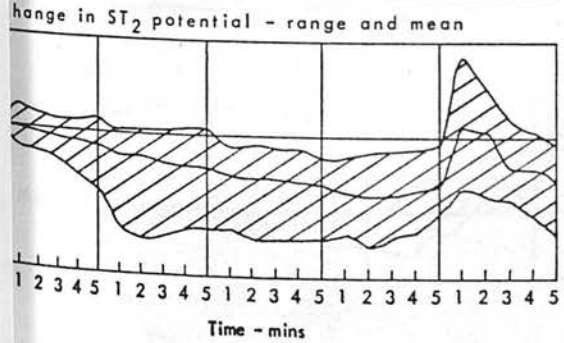


FIG. 2 Trained subjects. a (above): *range of ST_2 potentials.* b (below): *change in ST_2 potentials (range and mean) on exercise.*

ing the final minute of each work load. The expired gas from the Douglas bags was analysed for O_2 and Co_2 content using a paramagnetic (Servomax, Ltd) and Infra red (Beckman & Co., Ltd) analyser respectively. Details of the accuracy which might be expected from these methods have already been published (Davies and Shirling, 1967).

3 ECG leads

A single ECG lead was employed corresponding to position CR_5 (one electrode on the tip of the right shoulder and the other in the left anterior axillary line at the vertical height of the apex beat). The indifferent electrode was applied to the right chest. The electrodes used were small adhesive electrodes (Devices Ltd) which were applied to the skin after careful preparation by rubbing in electrode jelly. The electrodes were lightly taped to the skin and fine wires were employed which did not impede the movement of the subject during exercise.

4 ECG amplifier

This was a standard type and conformed to the specifications of the Ministry of Health as regards frequency response and time constant.

5 Tape recorder

The ECG signal was recorded continuously on magnetic tape at a speed of $1\frac{7}{8}$ in./sec. In each record 1 mV calibrations were included.

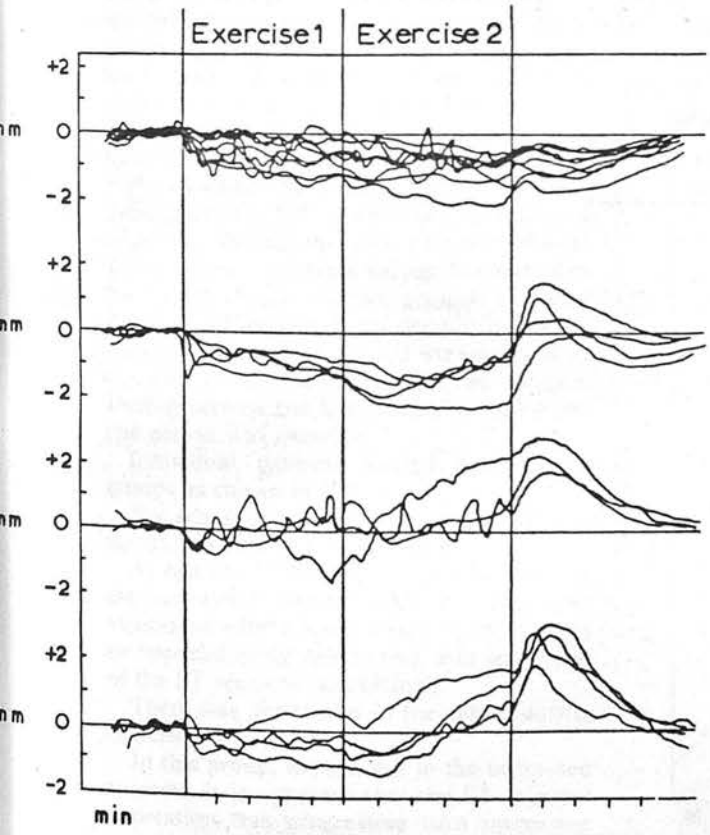
6 Computer

The customary method of studying ECG changes is to take two or more records at different times and compare them in terms of waveform. If changes are found, it is not known at what point they occurred, whether suddenly or gradually and whether they were associated with other events. This method of studying ECG changes may be called a time-sampling method. A repetitive temporal event like the waves of the ECG may be looked upon in another way, however; we may follow continuously a number of predetermined points on the waveform and record the way in which the potential at these points varies with time. For example we might record continuously the potential at a point 0·1 seconds after the onset of the R wave and follow changes in potential at this point over a period of time. This may be called the wave-sampling method.

TABLE 2 *Effect of exercise on ST segment voltage (mm)*

Untrained subjects (31)				
	ST_2	ST_2 *change*		
	Resting	*Light exercise*	*Severe exercise*	*Post-exercise*
ean	+0·31	−0·44	−0·34	+0·58
)	0·779	0·352	1·024	1·244
Em	0·014	0·099	0·184	0·223
		4·42	1·84	2·61
		<0·001	<0·100	<0·025

Trained subjects (12–17)						
pm/min	*Resting*	*300*	*600*	*900*	*1,200*	*Post-exercise*
ean	+0·68	−0·24	−0·64	−0·93	−0·94	+0·23
)	—	0·377	0·588	0·567	0·522	0·826
Em	—	0·091	0·143	0·142	0·151	0·229
		2·64	4·48	6·54	6·23	1·01
		<0·050	<0·001	<0·001	<0·001	n.s.



A computer based upon this principle has been designed (Neilson, Davies, and Kitchin, 1968). The computer performs the following functions:

a It reads the potential of each ECG cycle at a chosen number of points following the R wave, the reference potential being that 48 msec preceding the same R wave – that is, during the PQ interval.

b It records on any suitable recorder and at any desired paper speed the potential of these points over any period of time together with the instantaneous heart rate and does so as the ECG is being recorded.

c By replaying the tape of the ECG, further points on the waveform may be plotted so that a picture of the waveform may be filled in in as much detail as is required.

d Even using special electrodes and lead positions it is difficult to record, with conventional apparatus, a clear ECG signal during severe exercise. This noise is minimized by averaging a chosen number of succeeding complexes. This has the effect of greatly improving the signal to noise ratio.

In the present study the reference point was positioned 46 msec before the upstroke of the R wave and three sampling points on the ST segment were chosen at 20 msec intervals apart. The first point, ST_1 was selected after examination of the waveform of the ECG so as to be clear of the J point, and on the straight portion of the ST segment. Average positions after the onset of the R wave for points $ST_{1,2, \text{ and } 3}$ were 50 msec, 68 msec, and 86 msec.

7 Read out

The read out was on ECG paper, recorded at a speed of 1 cm/min. A 4-channel Devices recorder was used on one channel heart rate, and on the other three ST_1, ST_2, and ST_3 potentials were recorded.

8 Data analysis

The data was analysed in two ways: (1) in order to compare with previous studies, the behaviour of the ST segment during the last 1-min period of each phase of the study was recorded in terms of ST shift and slope; (2) in order to assess the time course of changes and the variability of changes during exercise, the average and range for 1-min intervals was measured throughout the study.

Results

1 Height of ST segment

Figures 1a and 2a show for all the untrained and trained subjects the range of values of ST_2 above or below the reference point of the PQ segment throughout the experiment.

FIG. 3 Untrained subjects: *Change in ST_2 potential during and after exercise – different patterns of response in four sub-groups of subjects.*

TABLE 3 *Effect of exercise on ST segment slope (mV/sec)*

jects (31)			
ST slope Resting	*Change in slope on exercise*		
	Light exercise	*Severe exercise*	*Post-exercise*
1·38	+0·98	+2·44	+2·71
1·440	0·844	1·410	1·460
0·026	0·151	0·254	0·362
	6·47	9·65	8·85
	<0·001	<0·001	<0·001

cts (12–17)					
ting	*300*	*600*	*900*	*1,200*	*Post-exercise*
	+0·88	+1·75	+2·83	+4·16	+4·88
	1·596	1·542	1·625	2·047	1·544
	0·387	0·374	0·406	0·591	0·428
	2·64	4·48	6·54	6·23	11·41
	<0·050	<0·001	<0·001	<0·001	<0·001

Figures 1b and 2b show the same data re-plotted giving the ST segment level during the control resting period the value 0. They therefore show the change in ST segment level during and after exercise. There is clearly a wide scatter, some subjects having ST depression of up to 1 mm, while others have ST elevation. (1 mm = 0·1 mV.)

Both trained and untrained groups show on average a fall in ST_2 on exercise followed by a large rise during the post-exercise minute. Table 2 shows the mean values, SD and SEm for ST_2 at rest in the two groups, and also the mean differences from the resting values (paired results) with graded exercise and at the end of the first post-exercise minute. During exercise the last minute of each exercise period was averaged.

Individual patterns tended to fall into groups as shown in Fig. 3.

The group of trained subjects differed from the untrained in the following ways.

At rest the ST segment was higher than in the untrained group (Table 2). This was associated with a lower mean heart rate (75 as opposed to 92 beats/min), and less slope of the ST segment (see below).

There was less scatter in the values during exercise.

In this group, though not in the untrained subjects, it is apparent that the ST segment depression was progressive with increasing severity of exercise.

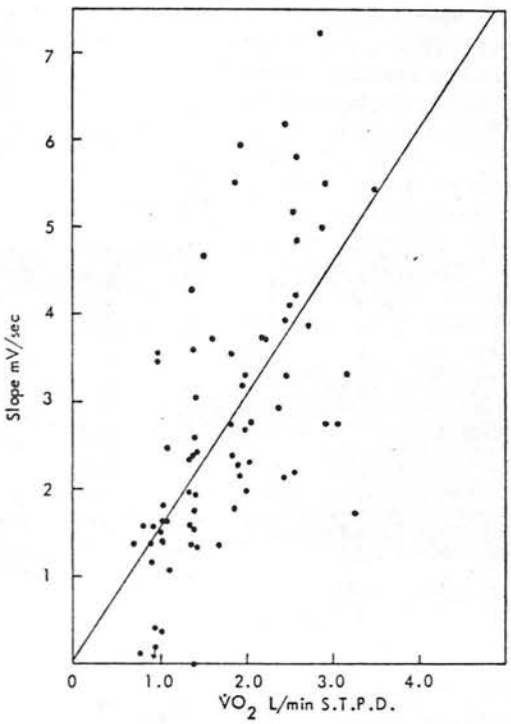


FIG. 4 *Change in ST slope on exercise plotted against oxygen consumption.*

FIG. 5 *Change in ST slope on exercise plotted against heart rate.*

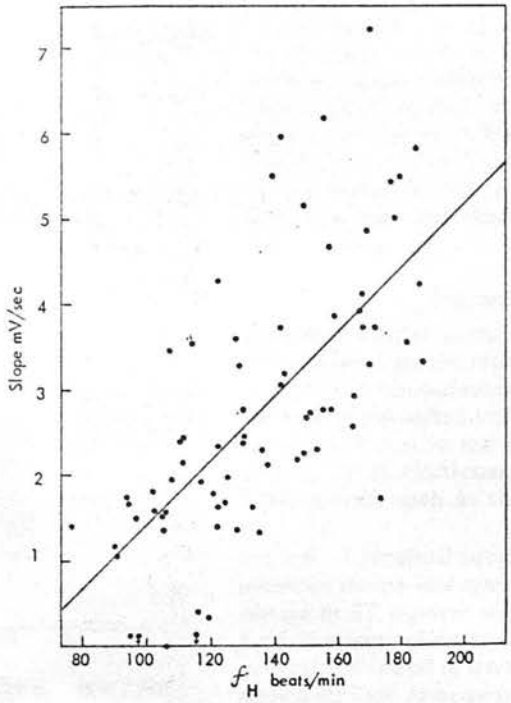
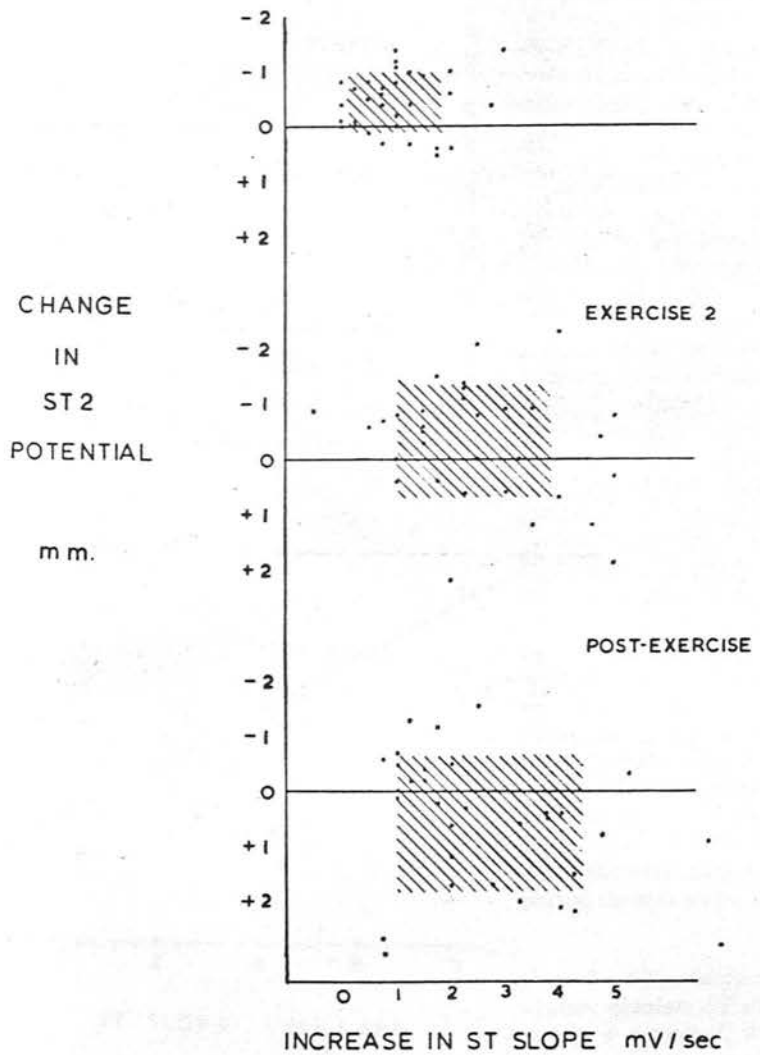


TABLE 4 *Correlation between ST_2 shift (mm) and change in ST slope (mV/sec) during and after exercise*

Work load	*Regression coeff. slope change/ST shift*	*Correlation coefficient r*
A *Untrained subjects*		
Light exercise	−0·10	−0·07
Severe exercise	+0·27	+0·19
Post-exercise	+0·46	+0·35
B *Trained subjects*		
300 kpm/min	+0·90	+0·21
600	+0·12	+0·05
900	−0·03	−0·01
1,200	+0·37	+0·09
Post-exercise	+0·32	+0·17



2 ST segment slope

Points ST_1 and ST_3, approximately 40 ms apart, were used to measure the slope of the ST segment, or rather of that portion of 70–110 msec after the R onset which is nearly rectilinear.

In both groups of subjects increasing exercise caused an increase in the slope of the ST segment. Cessation of exercise was followed by a further increase in slope (Table 3).

In the trained subjects the resting heart rate was lower and the slope of the ST segment more horizontal than in the untrained subjects. The relation of ST segment slope in the trained subjects to Vo_2 is shown in Fig. 4 ($r = 0{\cdot}732$ and $P = < 0{\cdot}001$) and to cardiac frequency in Fig. 5.

The correlation and regression coefficients for changes in ST segment and changes in slope are shown in Table 4.

3 Combined changes in ST segment height and slope

The combined changes in ST_2 and slope are shown for different grades of exercise in Figs. 6 and 7. The same general pattern is shown by both trained and untrained subjects – that is, with increasing intensity of exercise there is an increase in both slope and ST depression. During recovery, however, the slope is maintained or increased while the ST segment changes from depression to marked elevation.

In order to compare the data with those of McHenry *et al.* (1968), the ST segment response on exercise has been plotted against the absolute slope during severe exercise (for trained subjects) and exercise at 900 kpm/min (trained subjects). This is shown in Fig. 7. Four of the 31 untrained and four of the 15 trained subjects fell outside the normal limits for slope and depression proposed by these authors.

Discussion

Graded muscular exercise such as treadmill walking provides the most natural and realistic test of cardiopulmonary efficiency and in the case of suspected ischaemic heart disease a good provocative test for the development of pain or of electrocardiographic changes. Various tests such as the Master Sar

FIG. 6 Untrained subjects. *Change in ST potential during and after exercise related to change in ST segment slope. Average values for last minute of exercise in each person and at 1 min after end of exercise. The left point represents light exercise (exercise I).*

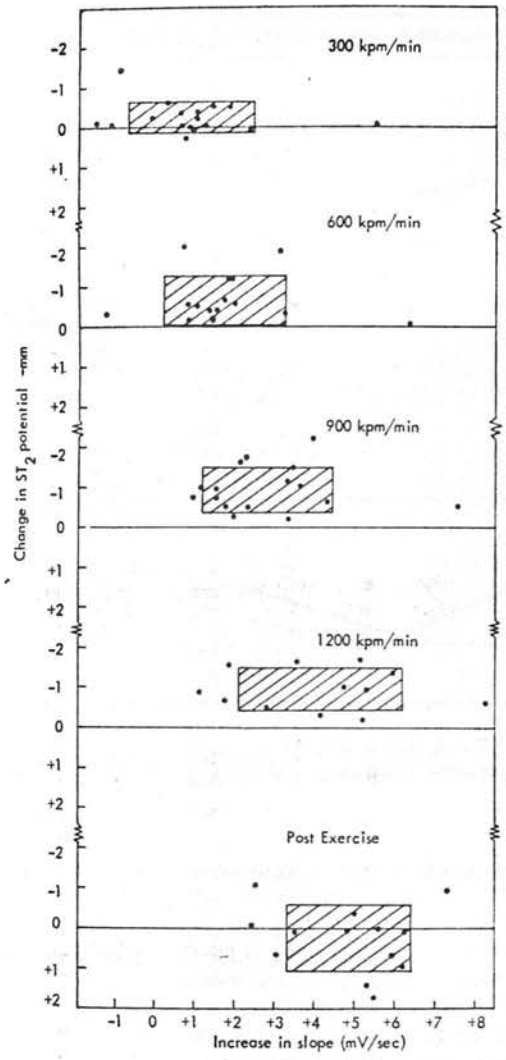


FIG. 7 Trained subjects. *Change in ST_2 and ST slope with increasing rate of exercise.*

(Master and Rosenfeld, 1961) which have been proposed have, however, shown a disappointing number of false positive (Friesinger *et al.*, 1965) and false negative (Sheffield, Holt, and Reeves, 1965) results. Modifications of the test – for example, using multiple ECG leads (Mason, Likar, Biern, and Ross, 1967) or using maximal exercise (Bellet and Roman, 1967) – improve the discrimination to some extent, but when an abnormal response is recorded in an apparently healthy subject there always remains a doubt as to its significance and whether the result is a false positive or represents a latent coronary ischaemic pattern. More elaborate tests such as those of Bruce, Mazzarella, Jordan, and Green (1966) have shown a high incidence of positive results in subjects without other evidence of heart disease, and this has been interpreted as suggesting that these individuals have latent coronary artery disease. (Mattingly, 1962; Rumball and Acheson, 1963; Robb and Marks, 1964; Bellet, Roman, Nichols, and Muller, 1967).

Some authors, however, believe that such patterns may occur without coronary artery disease in untrained subjects or in hyperventilation (Holmgren *et al.*, 1959; Friesinger *et al.*, 1965).

Our results show that there is a wide scatter of ST shift on exercise in untrained subjects, some changes falling well within the criteria accepted for ischaemic disease. These results conflict with the reports of Sheffield *et al.* (1965) and of Bellet and Roman (1967) who record that they have never seen such patterns in young normal subjects. The present technique has also permitted an unusually full study of the time course of these changes. Frequently they come on markedly at the start of exercise or at an increase in the level of exercise, at the time when the heart rate is increasing, and then tend to wear off as exercise continues. In other cases they gradually increase during exercise, as is the case with definitely proved cases of myocardial ischaemia. After the end of exercise there is almost always a rise in the ST segment with little further increase in slope while the heart rate is decreasing. During this period there is also a very large increase in the

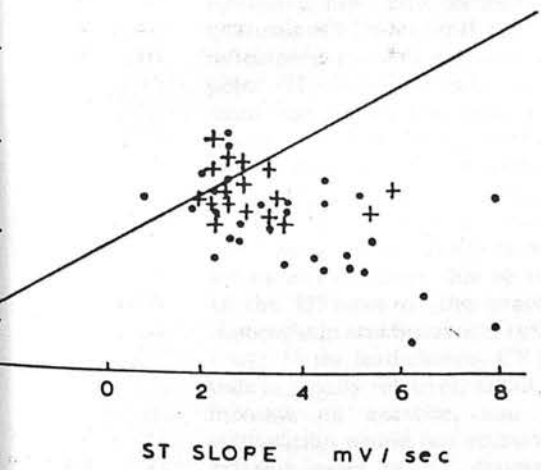


FIG. 8 *ST_2 change on exercise plotted against absolute ST slope (McHenry* et al., *1968). + = trained. ● = untrained subjects.*

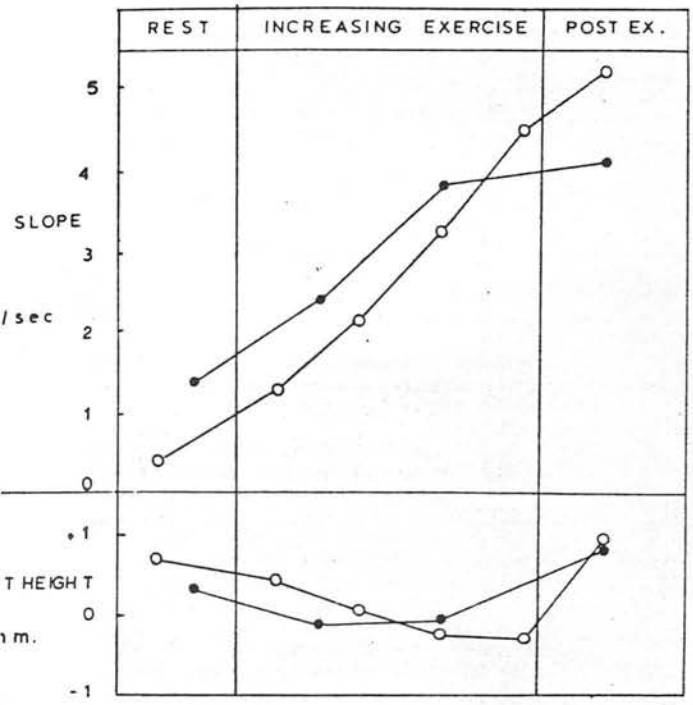


FIG. 9 *Mean values for ST slope and ST_2 height.* ● *= untrained.* ○ *= trained subjects.*

amplitude of the T waves, and the complex has the appearance of the T wave encroaching on the ST segment (Fig. 9).

The physiological interpretation of this sequence of changes must depend on an appraisal of how far they are predetermined by technical factors. In particular the choice of a reference point against which to measure ST segment amplitude presents difficulties at fast heart rates, since the proximity of the P wave to the preceding T wave obscures an isoelectric line. This difficulty is inherent in all forms of ECG exercise test. It is customary to employ the PQ segment as a reference point. The WHO criteria takes the PQ segment just before the onset of the Q wave (Rose and Blackburn, 1968). The present data were recorded using a point near the end of the PQ segment, well clear of the end of the P wave. Apparent ST depression at fast heart rates could equally well be accounted for by PQ elevation due to superimposition of the U wave of the preceding complex (Lepeschkin and Surawicz, 1958; Lepeschkin, 1965). In the lead chosen, CR5, the U amplitude is usually relatively small, though it may increase on exercise, and therefore this explanation would not account for the more extreme cases of ST depression. It does, however, represent a variable factor which is impossible to control. In ischaemic subjects, on the other hand, the U wave is frequently negative and this factor would tend to lessen the apparent ST depression at fast heart rates in this group of subjects.

It has been suggested on the basis of similar studies (McHenry *et al.*, 1968) that if in a group of subjects ST depression is plotted against ST slope on exercise, a line of separation of ischaemic from non-ischaemic cases can be obtained (Fig. 8). The present data do not fully agree with this, since eight cases (four trained and four untrained) fall outside the line of discrimination.

The post-exercise electrocardiogram (Master and Rosenfeld, 1961; Mattingly, 1962; Rumball and Acheson, 1963; Robb and Marks, 1964) is recorded at a time when the present data indicate that the normal ECG will have changed markedly from the pattern during exercise. The post-exercise response must therefore have a different significance. A positive result at this point must indicate a greater degree of ST depression relative to the normal. As a test of myocardial ischaemia it may therefore have greater significance than a similar degree of ST depression recorded during exercise.

The results obtained in trained subjects show a hitherto unreported range of amplitude change of the ST segment in normal individuals during graded physiological exercise. This is relevant to the choice of criteria of normality for exercise electrocardiography. While none of the subjects showed horizontal ST depression of more than 2 mm on or after exercise, a large number showed ST depression of 1–2 mm during exercise with upward sloping ST segments. These results obtained in trained young subjects do not differ from those obtained in untrained subjects, except that they show a more homogeneous pattern of response with less scatter of values at the various exercise levels. They also show a stepwise progression of changes with increasing grades of exercise, the ST_2 point following steadily and the slope of the ST segment increasing. This confirms the view that this type of response is a 'normal' one. It occurs in extremely fit, normal subjects and might be expected to occur at considerably lower levels of exercise in unfit subjects as found by Holmgren *et al.* (1959) and Friesinger *et al.* (1965). It has been proposed that such changes recorded in normal middle-aged individuals may indicate a risk of incipient coronary artery disease (Doan *et al.*, 1965). Our results would indicate that such changes should not be given this interpretation.

The computer used in this study was developed with the assistance of a grant from the Scottish Hospitals Endowments Research Trust. The British Heart Foundation gave a grant for the purchase of a tape recorder.

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CARDIO-RESPIRATORY RESPONSE TO EXERCISE IN NORMAL CHILDREN

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SUMMARY

1. The results of studies during simple progressive exercise to exhaustion and steady-state submaximal exercise in 117 boys and girls aged 6–16 years are presented.

2. In the simple progressive exercise test, the highest work load achieved and the submaximal heart rate were related to size and sex. The maximum heart rate and submaximal ventilation were largely independent of size and sex.

3. Steady-state exercise was performed at one-third and two-thirds of the maximum work load achieved in the simple progressive test. The Indirect (CO_2) Fick method was used to measure cardiac output.

4. At any given level of steady-state work, tidal volume, dead space, heart rate and stroke volume were closely related to size, with girls having higher heart rates and smaller stroke volumes than boys. Minute ventilation and cardiac output were virtually independent of size and sex. The cardiac output in children was the same as that in the adult for any given oxygen consumption. Blood lactate was related to size at any given work load, but was independent of size at any given fraction of the maximum working capacity.

The development of the Indirect (CO_2) Fick method for measuring cardiac output during exercise (Higgs, Clode, McHardy, Jones & Campbell, 1967) and of mathematical techniques for analysis of the results (McHardy, Jones & Campbell, 1967; Godfrey, 1970a) have made it possible to study exercise physiology with minimal inconvenience to the patient. Standard techniques for measurement of cardiac response to exercise by either the Direct (O_2) Fick method or the dye-dilution method require cardiac and arterial catheterization and are difficult to justify for the study of normal children. The Indirect (CO_2) Fick method which uses a rebreathing technique for estimating mixed venous P_{CO_2} (Jones, Campbell, McHardy, Higgs & Clode, 1967) and either end-tidal P_{CO_2} or ear-lobe blood P_{CO_2} (Godfrey, Wozniak, Courtenay Evans & Samuels, 1971) is safe, reliable and acceptable to either healthy or sick children (Godfrey, 1970b).

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ous information about exercise physiology in children has generally been limited to surement of physical working capacity, heart rate and ventilation (Astrand, 1952; n, 1956; Adams, Linde & Miyake, 1961). Gadhoke & Jones (1969) succeeded in forty normal boys aged 11–14 by using some of the techniques outlined above. To the results of studies in children with heart or lung disease a much wider range of values was needed and we therefore decided to study children of both sexes aged from years.

SUBJECTS AND METHODS

were made on fifty-seven boys and sixty girls evenly distributed in age, height and Table 1). All children were volunteers from local schools and fully informed consent ined in writing from their parents. The children were examined clinically on the day t and signs of heart, lung or neuromuscular disease were not detected. The studies

TABLE 1. Distribution of subjects by age, height and weight

	Age group (years)										
	6·0–6·9	7·0–7·9	8·0–8·9	9·0–9·9	10·0–10·9	11·0–11·9	12·0–12·9	13·0–13·9	14·0–14·9	15·0–15·9	Total
	5	6	6	5	7	5	7	6	5	5	57
	5	6	6	6	8	6	6	6	6	5	60

	Height group (cm)							
	110·0–119·9	120·0–129·9	130·0–139·9	140·0–149·9	150·0–159·9	160·0–169·9	170·0–179·9	180·0–189·9
lo. of boys	6	8	13	10	9	3	5	3
lo. of girls	6	9	11	13	11	10	0	0

	Weight group (kg)									
	15·0–19·9	20·0–24·9	25·0–29·9	30·0–34·9	35·0–39·9	40·0–44·9	45·0–49·9	50·0–54·9	55·0–59·9	60·0–76·5
s	3	9	15	2	9	4	6	2	4	3
	5	7	10	10	4	5	3	8	5	3

ned during the morning and afternoon after light meals and at least 1 h was left s in any one subject.

of exercise test were performed by each child on a cycle ergometer (Lode) in osition.

progressive exercise

is test the work load on the ergometer was increased every minute and the electro-
raph and inspired ventilation were recorded continuously on a chart recorder (Mingo-
). Ventilation was measured by using a valve box of low resistance and dead space
for older children, 30 ml for the younger), with a gas meter (Parkinson–Cowan C.D.4)
in the inspiratory line. The increments of work depended on the size of the child; 10 W
ldren less than 120 cm in height, 15 W for children from 120 to 150 cm, and 20 W
ldren over 150 cm. The child was encouraged to persevere until exhaustion. By using
ents of work related to size meant that most children exercised progressively harder
out 6–8 min. The highest work load completed ($W_{max.}$) was noted and the heart rate
ntilation for every load were calculated.

-state exercise

r a complete rest of at least 60 min a test was performed in which each child was studied
and then at one-third and two-thirds of the previously determined $W_{max.}$ under steady
ions. The work loads were performed without any rest between them. Expired gas was
l through a large Tissot spirometer and analysed continuously for O_2 and CO_2. After
min of exercise, when the heart rate, ventilation and expired gas concentrations were
o be steady from the continuous record (i.e. varying by less than 5%), a collection of
d gas was made over at least 1 min in the spirometer and was immediately analysed.
ıple of arterialized ear-lobe blood was obtained by the method of Godfrey *et al.* (1971)
during the collection or immediately afterwards in the more timid children. This was
ısed to obtain values for the normal physiological dead space (see below). The oxygenated
l venous P_{CO_2} ($\bar{P}vCO_2$) was measured by a rebreathing method (Jones *et al.*, 1967) in
n the subject was given an appropriate volume and concentration of CO_2 in O_2 to re-
he for 12–15 s. The end-tidal P_{CO_2} was recorded continouusly to identify the plateau
senting equilibration between mixed venous P_{CO_2}, alveolar P_{CO_2} and rebreathing bag
. If a perfect plateau (i.e. one which appeared within 3 or 4 s and broke with recirculation
) s) was not obtained, the extrapolation procedure described by Denison, Edwards, Jones
ope (1969) was used. No correction was applied for the difference between the plateau
2 and that in blood arriving at the lungs or leaving them during the equilibrium described
ones *et al.* (1967) and Jones, Campbell, Edwards & Wilkoff (1969).

lyses and calculations

pired O_2 was measured with a paramagnetic analyser (Servomex OA150) modified to
a full-scale deflection on the recorder over an appropriately restricted range. Expired and
tidal CO_2 was measured with an i.r. analyser (URAS-4) which also wrote on the recorder.
analysers were calibrated with four gas mixtures spanning the operative range after every
or three studies. Ear-lobe blood was analysed immediately for blood gases by using
ıweiler microelectrodes calibrated with gases and buffers for every sample. Ear-lobe blood
ate was determined by the method of Gerken (1960). All calibrating gases were analysed
mically with a Lloyd-Haldane apparatus. All ventilations are expressed at B.T.P.S. and all
consumptions at S.T.P.D.

The results were calculated with the help of a digital computer program (Godfrey, 1970a).
the present study cardiac output was calculated by using the arterial P_{CO_2} implied by

assuming that the subject had a normal physiological dead space. The values for the dead space in normal children were determined during the study and agreed closely with those based on weight (Radford, 1954). The use of an assumed normal dead space to calculate arterial P_{CO_2} and subsequently cardiac output has been discussed by Godfrey & Davies (1970).

Preliminary analysis suggested that height was the most useful index of body size for almost all comparisons and the data have therefore been presented in relation to sex and height unless otherwise stated. Since the Indirect (CO_2) Fick method for cardiac output is unreliable at rest (Godfrey & Davies, 1970) regressions were based on work only. Some data on rest are included as stated in the text.

RESULTS

The individual results for body size and the highest work levels completed in the progressive exercise test are presented in *Clinical Science* Table 40/2, which has been deposited with the Librarian of the Royal Society of Medicine, London, W.1. The individual results for steady-state exercise tests have been deposited as *Clinical Science* Table 40/3.

Simple progressive exercise

The highest work load completed by the child ($W_{max.}$) was found to depend significantly on size and sex, boys achieving higher loads than girls of similar height (Fig. 1, Table 2).

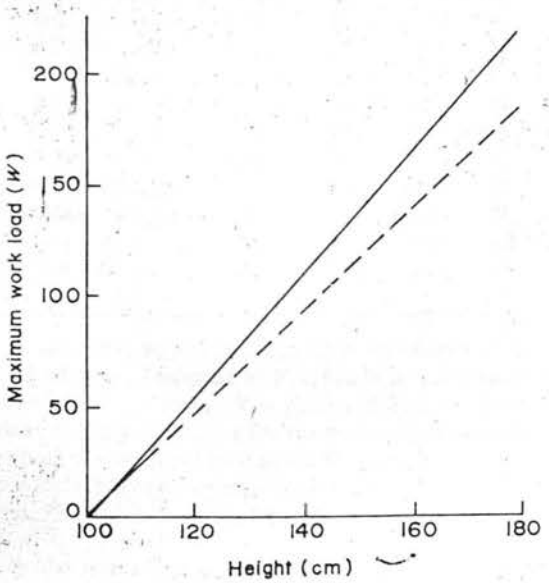

FIG. 1. Highest work load completed ($W_{max.}$) in simple progressive exercise in relation to height and sex. – – – –, Girls (SE Y = 17); ——, boys (SE Y = 19).

The same applied to the highest ventilation achieved. The highest heart rate achieved was 196±13 (SD) beats/min in girls and 195±13 (SD) beats/min in boys and was independent of height.

At all submaximal work levels the heart rate was significantly related to work load, height and sex, with girls having higher heart rates than boys. Smaller children had higher heart rates

vel than larger children and their increase in heart rate for unit increase in work
er. This meant that multiple linear regression could not adequately express the
ing the children by height and performing a simple linear regression for each
d the analysis, but the height range of the groups had to be large to get a
er of points. A practical compromise was obtained by performing the multiple
ulse on logarithm of work and height (Table 2) which adequately expressed the

TABLE 2. Regression equations for all results

	Y	B_1	X_1	B_2	X_2	M	SE Y	r	n
rogressive exercise									
s	$W_{max.}$	2·87	Height	—	—	−291	19	0·94	55
s	$W_{max.}$	2·38	Height	—	—	−238	17	0·91	58
s	$\dot{V}_E$	0·386	W	−0·094	Height	22·9	8·4	0·92	406
s	$\dot{V}_E$	0·402	W	−0·117	Height	23·8	6·1	0·93	365
s	Pulse	87·6	log$W$	−1·073	Height	162	16	0·85	410
s	Pulse	84·2	log$W$	−1·171	Height	191	15	0·85	351
ite exercise									
	$\dot{V}_{O_2}$	11·89	W	—	—	329	127	0·96	102
	$\dot{V}_{O_2}$	11·20	W	1·701	Height	109	94	0·96	98
	$\dot{V}_E$	0·0305	$\dot{V}_{O_2}$	−0·117	Height	17·0	5·0	0·93	103
	$\dot{V}_E$	0·0370	$\dot{V}_{O_2}$	−0·211	Height	25·7	4·5	0·92	98
	V_T	0·465	$\dot{V}_{O_2}$	8·03	Height	−826	159	0·91	103
	V_T	0·490	$\dot{V}_{O_2}$	6·09	Height	−577	111	0·91	98
	V_D	1·54	Weight	0·049	V_T	2	22	0·78	40
	$\dot{Q}$	0·0057	$\dot{V}_{O_2}$	0·022	Height	0·71	0·92	0·95	87
s	$\dot{Q}$	0·0056	$\dot{V}_{O_2}$	0·021	Height	1·02	0·74	0·94	74
	Pulse	159·1	log$\dot{V}_{O_2}$	−1·204	Height	−143	18	0·71	103
s	Pulse	173·0	log$\dot{V}_{O_2}$	−1·395	Height	−140	14	0·81	98
	SV	0·0114	$\dot{V}_{O_2}$	0·636	Height	− 41	10	0·85	87
s	SV	—	—	0·645	Height	− 37	7	0·81	75
	$P\dot{V}CO_2$	0·0098	$\dot{V}_{O_2}$	—	—	52·9	4·2	0·70	89
s	$P\dot{V}CO_2$	0·0142	$\dot{V}_{O_2}$	—	—	46·9	5·1	0·64	79

nt variable; X_1 and X_2, independent variables; B_1 and B_2, regression coefficients of X_1 tively; M, constant term; SE Y, standard error of estimate of Y; r, (multiple) correlation degrees of freedom. The equations are of the form: $Y = B_1X_1 + B_2X_2 + M \pm SEY$.
ue is given for B or X, the equation was only significant for the remaining parameter
/ise all equations are significant for both X_1 and X_2 at least at the 5% level.
for Y and X are those given in the text. Other abbreviations and units are as follows:
eight (kg), work (W), $\dot{V}_{O_2}$ (ml/min), $\dot{V}_E$ (l/min), V_T (ml), V_D (ml), $\dot{Q}$ (l/min), SV (ml),
n), $P\dot{V}CO_2$ (mmHg).
f independent variables covered by the regressions were: height 113–182 cm, weight
: 50–260 W, $\dot{V}_{O_2}$ 320–2420 ml/min.

submaximal work levels ($\dot{V}_E$) was highly significantly related to work load of sex (Table 2). Although it was also related to height, the variation over the children studied was less than twice the SE of the estimate.

Steady-state exercise

All steady-state results refer to submaximal work. The most important results are illustrated in the figures and all the regression equations are given in Table 2.

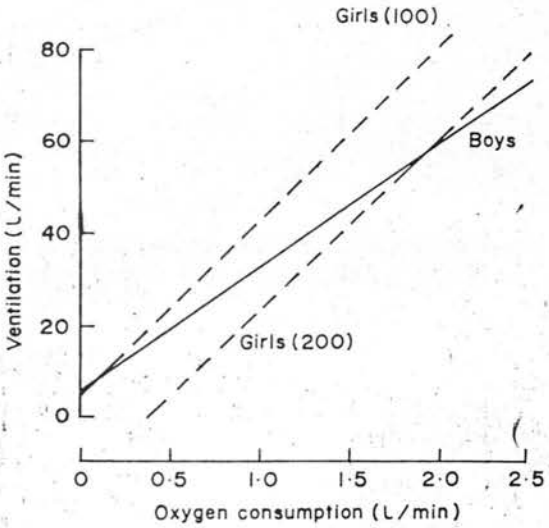

FIG. 2. Ventilation during steady-state exercise in relation to $\dot{V}O_2$ and to height in girls. The lines for theoretical heights of 100 cm and 200 cm are shown. The effect of height was not significant in boys. Boys, SE Y = 5·1; girls, SE Y = 4·5.

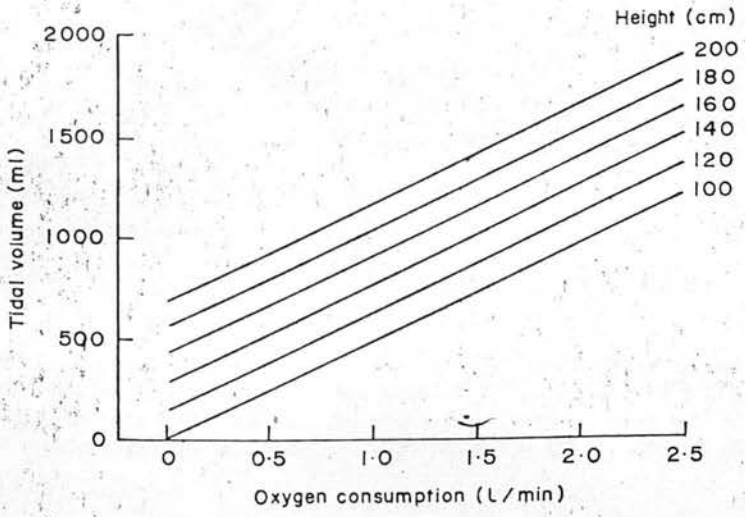

FIG. 3. Tidal volume during steady-state exercise in relation to $\dot{V}O_2$ and height. Small differences due to sex have been omitted. Boys and girls, SE Y = 135.

Oxygen consumption ($\dot{V}O_2$). This was significantly related to physical work with no significant sex or size difference over the operative range, although it tended to be lower in smaller girls. A regression was calculated for all other measurements on $\dot{V}O_2$ except as described.

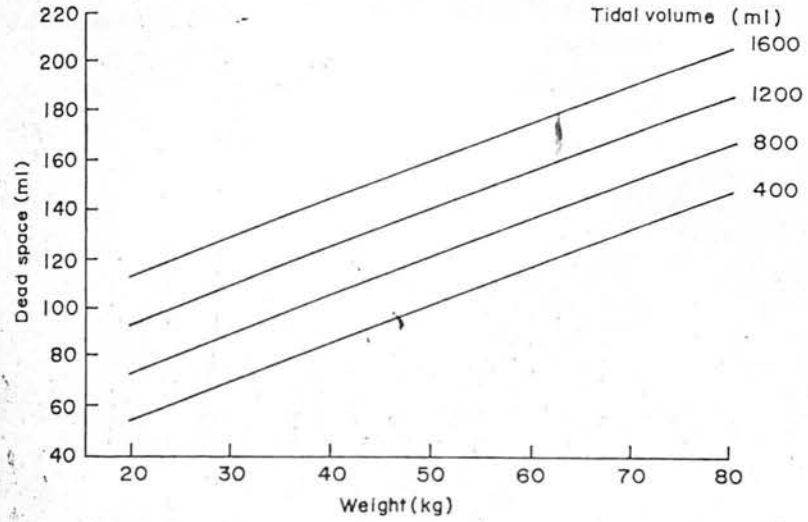

FIG. 4. Physiological dead-space during steady-state exercise in relation to weight and tidal volume for boys and girls together (see the text). Boys and girls, SE $Y = 22$.

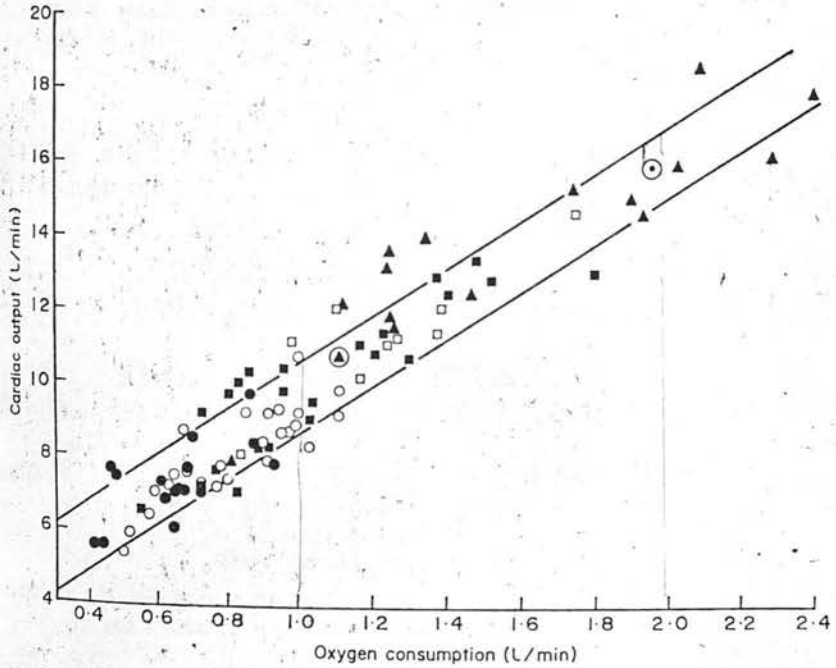

FIG. 5. Cardiac output during steady-state exercise in relation to $\dot{V}O_2$ for all boys in various height groups. The lines represent ± 1 SE Y about the regression line. The regression line for girls was not significantly different. The circles represent the results of the study in adult men by Bevegard *et al.* (1960). Height range (cm): ●, 110–124; ○, 125–139; ■, 140–154; □, 155–169; ▲, 170–185.

Ventilation ($\dot{V}_E$). This was significantly related to $\dot{V}O_2$ in all groups with no size difference in boys over the operative range. There was a small but significant size effect in girls (Fig. 2).

Tidal volume (V_T). This was significantly related to $\dot{V}O_2$ and to size, being larger at any given

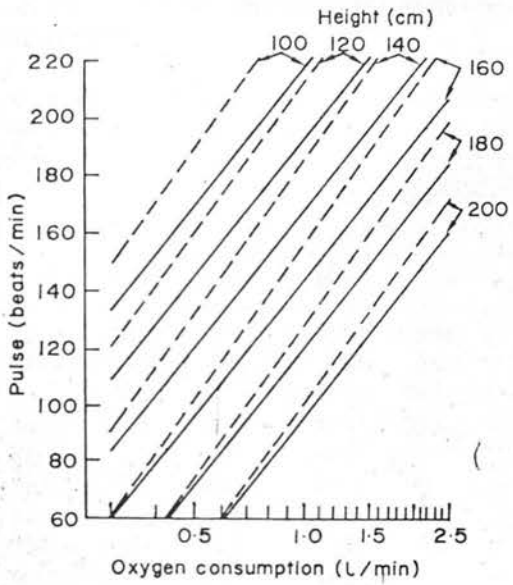

FIG. 6. Heart rate during steady-state exercise in relation to $\dot{V}O_2$, height and sex. – – – –, Girls (SEY = 14); ——, boys (SEY = 18).

1·2

. 7. The scale for arterial PCO_2 is wrong. The
orrect values for all three parameters are as follows.

	Rest			⅓ Max			⅔ Max		
♂	.970	(.017)	45	.967	(.011)	54	1.082	(.011)	52
♀	.959	(.019)	45	.961	(.013)	52	1.074	(.011)	49
♂	33.6	(.7)	36	37.4	(.5)	48	35.0	(.5)	42
♀	32.1	(.9)	32	35.8	(.7)	46	33.9	(.7)	33
♂	1.2	(.1)	35	2.1	(.2)	42	4.9	(.3)	40
♀	1.0	(.1)	25	2.7	(.2)	37	5.3	(.3)	34
	Rest	Work1	Work2				Rest	Work1	Work2

FIG. 7. Steady-state respiratory exchange ratio (R), arterialized ear-lobe blood lactate concentration and arterial P_{CO_2} at rest, one-third and two-thirds of maximum working capacity. ●, Boys; ○, girls. The bar includes ±SEM about the mean value shown by the symbol.

$\dot{V}O_2$ value in taller children. There was no significant sex difference. It was found in this instance that multiple linear regression expressed the results best. The results are illustrated in Fig. 3. There was no consistent relationship between V_T and $\dot{V}_E$.

Physiological dead space (V_D). This was calculated from the Bohr equation by using both arterialized ear-lobe P_{CO_2} and end-tidal P_{CO_2} as alternative estimates of arterial P_{CO_2}. In forty-one technically satisfactory studies, each member of the pair of results for dead space lay within ±10% of their mean value. A multiple-regression analysis of these results showed that V_D was significantly related to weight (more than to height) and to tidal volume (more than to work level). There were not enough studies to enable any conclusions to be made about sex differences. The results are illustrated in Fig. 4.

TABLE 3. Comparison of parameters at rest, one-third and two-thirds of maximum working capacity; for explanation of calculated rise in blood lactate concentration see the text

		Rest		First work load		Second work load	
		Boys	Girls	Boys	Girls	Boys	Girls
R	Mean	0·97	0·96	0·97	0·96	1·08	1·07
	SEM	0·02	0·02	0·01	0·01	0·01	0·01
	n	45	45	54	52	52	49
P_{a,CO_2} (mmHg)	Mean	33·6	32·1	37·4	35·8	35·0	33·9
	SEM	0·7	0·9	0·5	0·7	0·5	0·7
	n	36	32	48	46	42	33
Measured concn. of lactate (mmol/l)	Mean	1·2	1·0	2·7	2·7	4·9	5·3
	SEM	0·1	0·1	0·2	0·2	0·3	0·3
	n	35	25	42	37	40	34
Calculated rise in lactate concn. (mmol/l)	Mean	—	—	2·0	1·7	3·3	2·8
	SEM	—	—	0·2	0·1	0·2	0·2
	n	—	—	46	42	39	32

Cardiac output ($\dot{Q}$). This was significantly related to $\dot{V}_{O_2}$ with no sex differences. The influence of height, though significant, was small over the operative range (Fig. 5).

Heart rate (pulse). The heart rate was significantly related to $\dot{V}_{O_2}$ and was higher for smaller children and girls at any one work level. As in the simple progressive test it was found that the multiple regression of heart rate on the logarithm of $\dot{V}_{O_2}$ and height fitted the data best (Fig. 6).

Stroke volume (SV). Stroke volume was not significantly related to $\dot{V}_{O_2}$ in girls but there was a small, significant increase with $\dot{V}_{O_2}$ in boys (Table 2). There was a significant increase of SV with height in both sexes but girls had a lower SV value than boys of equivalent height.

Mixed venous P_{CO_2} ($P\bar{v}_{CO_2}$). This was significantly related to $\dot{V}_{O_2}$. There was no height effect but the regression lines for the sexes were slightly different.

Certain measurements were not significantly correlated with $\dot{V}_{O_2}$ or height but seemed to be related more to the relative work, i.e. whether it was rest, one-third or two-thirds of W_{max}. (Fig. 7, Table 3).

Respiratory-exchange ratio (R). This was moderately high at rest and did not change at the first work load. It increased significantly from the first to second work load. There was no sex difference.

Arterial P_{CO_2} (Pa,CO_2) was calculated from the Bohr equation for all studies in all subjects assuming a normal dead-space and correcting for instrument dead-space. It was rather variable at all levels but rose significantly from rest to the first work level and fell significantly from the first to the second level. The Pa,CO_2 was higher in boys at every level, but this was only significant at the first work load.

Blood lactate. The concentration of lactate in the blood rose very significantly from rest to the first work load and from the first to the second work load. Differences between the sexes were not significant. The increases in blood lactate concentration were calculated theoretically from a CO_2 balance equation (Clode & Campbell, 1969) and they are also given in Table 3. The calculated rises were virtually identical with the measured rises at all work levels for girls but were rather higher than the measured rises in boys, especially from the first to the second work load.

DISCUSSION

Details of the simpler measurements contained in this report such as pulse, ventilation and gas exchange are readily available for children (Robinson, 1938; Bengtsson, 1956; Astrand, 1952; Cumming & Friesen, 1967). However, information is not available about cardiac output and stroke volume on exercise, in relation to size over a wide range. This is because the standard techniques of cardiac catheterization are unacceptable for the study of normal children. The object of the present investigation was to provide indices of normal function in children, related to size where relevant, for clinical use. Equations and graphs have purposely been kept as simple as possible for this reason.

The methods used in this study follow the techniques developed by Campbell and his group (Higgs *et al.*, 1967; McHardy *et al.*, 1967; Jones *et al.*, 1967) and they have been fully discussed by those authors. The procedure has been modified slightly for use in children, and in particular we have developed the concept of using an assumed normal dead-space to calculate Pa,CO_2 (Godfrey & Davies, 1970). The reliability of determining cardiac output by the Indirect (CO_2) Fick method, compared with the Direct (O_2) Fick or Dye Dilution methods, has been shown in studies in adults (Higgs *et al.*, 1967; Ferguson, Faulkner, Julius & Conway, 1968; Muiesan, Sorbini, Solinas, Grassi, Casucci & Petz, 1968; Denison *et al.*, 1969) but not in children.

Little technical difficulty was encountered in obtaining the heart rate, ventilation and gas exchange on exercise in most of the children, but we failed to obtain reliable results in 7% of work levels in boys and 16% in girls (each child performing two work levels). By using rigid criteria for acceptability of the plateau in measuring mixed venous P_{CO_2} or in extrapolating the value (Jones *et al.*, 1967; Denison *et al.*, 1969) a total of 14% of work loads in boys and 20% in girls could not be used. However, at least one level of exercise was satisfactorily completed (including the measurement of cardiac output) in 91% of boys and 90% of girls.

Simple progressive exercise

By grading the work increments according to size most children, apart from the very smallest and the very largest, completed 6–8 min of progressive exercise. This test was simple to perform and provided information of clinical value on effort tolerance (the maximum load achieved) and on the pulse/work and inspired ventilation/work relationship at submaximal work levels. These values are often abnormal in heart or lung disease (Godfrey, 1970b).

Most of the children exercised up to or close to their limit in this test with a mean maximum

ite of 196. Astrand (1952) reported maximum heart rates of 202–211 in Swedish children
was in steady-state exercise and higher than in most other series. Wilmore & Sigerseth
eported maximum heart rates of 191–197 in girls aged 7–13 by using a similar technique
though the work increments used were 150 kpm/min (24·5 W) independent of size. In
l context therefore, if a child stops exercising with a heart rate below 190, it is highly
ve of an abnormality in the cardio-respiratory system unless he is not trying; in that
pulse/work and ventilation/work relationship at submaximal levels should be normal.
results obtained in the simple progressive exercise test agree closely with those reported
er workers (Wilmore & Sigerseth, 1967; Gadhoke & Jones, 1969). Thus it was found
e maximum heart rate and the submaximal ventilation/work relationship were largely
ndent of size, whereas the maximum work (and ventilation) and the submaximal pulse/
relationship was size dependent. Except in the very youngest children, boys could
lly achieve more work than girls and had lower pulse-rates for any given work. This
s somewhat with the finding of Astrand (1952) that most indices of physical fitness in
ly began to lag behind those of boys after the age of 12–13.

state exercise.

termining the working capacity in the simple progressive-exercise test it was possible
nt each child with two work loads, similar in a relative sense, for steady-state exercise.
ery difficult to obtain reliable gas collections at rest in many of the children but they
lways settled down as soon as exercise began. For this reason we have concentrated
nore reliable exercise studies.

lation and heart rate. As in the simple progressive-exercise test our results for expired
ion, heart rate and also for gas exchange agree with previously published data (Bengts-
56; Gadhoke & Jones, 1969), though direct comparisons of results are somewhat
owing to differences in the mode of presentation. Like them we found that tidal
was related to size at any given value of oxygen consumption, but size had less influence
l ventilation. Compared with the simple progressive-exercise test, heart rate and
ion were 5–10% higher in the steady-state test. This can be explained by the relative
y state existing in the simple progressive test.

ac output and stroke volume. One object of this study was to obtain reliable data on
output over a wide range of size in normal boys and girls. In a previous publication
y & Davies, 1970) it was shown that the cardiac output in normal subjects was best
d by using an assumed normal dead-space. This is perhaps more useful in children
lts because the dead-space changes considerably with size and can be predicted with
tainty (Fig. 4). There is very little information about the values for dead space in
other than in the neonatal period, except for that of Robinson (1938), in which he
an apparatus dead-space of 100 ml (Radford, 1954). Levinson (personal communica-
provided us with values of dead space at rest in normal children, calculated from
neasured arterial P_{CO_2}, which closely agree with results given here and with the
tions of Radford (1954). Tenney & Bartlett (1967) predicted from comparative
at dead space should be related to body weight.

ly study known to the authors of cardiac output in normal children during exercise
Gadhoke & Jones (1969) which was limited to boys of a narrow size range. There is
ble information about the cardiac output in relation to work in normal adults, that

of Bevegard, Holmgren & Jonsson (1960) being typical of many others. The results of their studies in adults have been included in Fig. 5 which shows that there was no significant difference between the present results and their findings in adults. The values of Gadhoke & Jones (1969) were rather higher than ours at lower values of oxygen consumption, presumably because they applied a 'downstream correction' to the mixed venous $P\bar{v}CO_2$ (Jones *et al.*, 1969), for the observed difference between the alveolar gas PCO_2 and the PCO_2 in the blood leaving the lungs during the rebreathing equilibrium plateau. This results in a smaller veno-arterial PCO_2 difference which is proportionately more significant at lower work levels. We have not applied this correction because its significance is in doubt. Thus Denison *et al.* (1969) found that cardiac output based on the rebreathing $P\bar{v}CO_2$ agreed with that based on rebreathing mixed venous PO_2 ($P\bar{v}O_2$), directly measured $P\bar{v}O_2$ and with the predicted cardiac output based on O_2 consumption. The result calculated from directly measured pulmonary arterial PCO_2 ('upstream') gave results that were too high.

Stroke volume was only a little influenced by work in the present study but it was related to size (Table 2). This agrees with previous findings that the stroke volume increases from rest to exercise but not much between exercise loads (Bevegard *et al.*, 1960). Unfortunately the results on cardiac output at rest were not reliable enough to calculate resting stroke volumes for comparison.

Blood lactate concentration. The method of grading exercise has allowed a comparison of lactate concentrations to be made; this was relatively unaffected by size. From Table 3 it can be seen that although there were significant rises from rest to work and from the first level to the second, the variation within any one work level was small. This suggests that the level of work at which lactate is produced increases as the child grows, and on a relative scale at submaximal levels of work small children produce no more lactate than larger children.

The increase in lactate was also reflected in the fall of Pa,CO_2 which occurred from the first to the second work load, and for the corresponding rise in R (Fig. 7). The rather low resting value for Pa,CO_2 reflects the difficulty in obtaining reliable resting studies in children. The relatively good agreement between the observed lactate rises and those predicted from the CO_2 balance equations (Clode & Campbell, 1969) means that this prediction has considerable practical value in reflecting the degree of anaerobic metabolism.

It is concluded that as children grow there is little sex difference in cardiorespiratory performance under the age of 16, apart from the slightly greater maximum work load achieved by boys. Growth has little effect on the relationship between O_2 consumption, total ventilation or cardiac output and the work level. Tidal volume, dead space, pulse rate and stroke volume are closely related to size at any level of work. Moderate alveolar hyperventilation is common in this type of exercise. Anaerobic metabolism, although related to size at any work level, is independent of size at any given fraction of the exercise capacity of the child.

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The directly measured maximum aerobic power ($\dot{V}O_{2max.}$) is now widely accepted as an international reference standard of physical working capacity (P.W.C.) in man. Various methods for measuring this have recently been compared (Shephard, Allen, Benade, Davies, di Prampero, Hedman, Merriman, Myhre & Simmons, 1968) and procedures agreed on for laboratory studies. However, direct measurement of $\dot{V}O_{2max.}$ still poses certain technical problems and the procedures involved cannot be applied to all segments of the population. This is particularly important when one wishes to consider community studies of P.W.C. in the clinical situation. There is still a great need for physiologically acceptable secondary standards based on data collected from submaximal levels of effort.

From the physiological viewpoint the $\dot{V}O_{2max.}$ is a measure of the integrated performance of the various systems that are responsible for transporting oxygen from the outside air to its site of utilization within the working muscles. These systems form the links in the overall O_2-transporting chain. They may be considered for convenience under three main headings: muscle tissue, pulmonary ventilation and blood circulation; and measured in terms of their functional and dimensional components (Holmgren & Åstrand, 1966) as shown in Table 1.

TABLE 1. Components of the O_2-transporting system

Aspect	Component	
	Dimensional	Functional
Lungs	Forced vital capacity (FVC)	Forced expiratory volume (FEV) maximum ventilation ($\dot{V}_{E_{max.}}$) ventilation at oxygen intake 1·5 l/min ($\dot{V}_E$, 1·5)
Circulation	Blood volume (BV) Total body haemoglobin (THb) Heart volume (HV)	Cardiac frequency at oxygen intake 1·5 l/min (fh, 1·5) maximum cardiac frequency ($fh_{max.}$)
Tissues	Weight, height, total muscle estimated from soft tissue X-ray and total body potassium (^{40}K), thigh muscle (TM), calf muscle (CM), subcutaneous fat estimated from soft tissue X-ray	
Overall	Maximum oxygen intake ($\dot{V}O_{2max.}$) maximum blood lactic and ($LA_{max.}$) blood lactic and at an oxygen intake of 1·5 l/min (LA, 1·5)	

The aim of the present investigation is to confirm and extend previous work from this laboratory on young men and women (Cotes, Davies, Edholm, Healy & Tanner, 1969) by analysing the functional and dimensional components of the O_2-transporting system in eighty healthy male subjects (ages 20–50 years) during work on an upright bicycle ergometer in relation to age, and to the indirect measurement of maximum aerobic power.

METHODS

The subjects were eighty healthy men aged 20–50 years. They were volunteers from a population of Royal Naval officers and men stationed at H.M.S. *Victory*, R.N. Barracks, Portsmouth. Their physical and anthropometric details are given in Table 2. Each subject was required to attend the laboratory on three different occasions. On the first visit to the laboratory a general

medical examination was carried out. On the second visit measurements of the responses to submaximal and maximal exercise, standard anthropometry (including soft-tissue radiographs), heart volume and lung function were made. On the final visit whole-body potassium, blood volume and total-body haemoglobin were determined. On each occasion the subjects were allowed to rest for 15 min before measurements were taken, but no attempt was made to obtain basal conditions.

The subjects were always studied in pairs and spent a total of 8 h in the laboratory being measured. All the subjects were volunteers and there was absolutely no form of compulsion to take part in the study. They appeared interested in the investigation and only one subject (because of a ruptured achilles tendon) failed to complete all tests.

The methods for the measurements of the variables shown in Table 1 have already been published by various workers and the reader is referred to the references given in parentheses after each procedure for more precise details. Only brief outlines of the methods involved will be given here.

Forced vital capacity (FVC) and forced expiratory volume in 1 s ($FEV_{1\cdot0}$) were determined by using a dry gas spirometer (Cotes, 1966). Blood volume (BV) was determined by an intravenous infection of ^{131}I-labelled albumin (Schultz, Hammarsten, Heller & Ebert, 1953). Total-body haemoglobin (THb) was derived from BV and measured haemoglobin (Hb) concentration (g/100 ml) of blood (Kjellberg, Rudhe & Sjöstrand, 1950). Heart volume (HV) was measured roentgenographically at rest in the prone position (Reindell, Kirhoff, Musshoff & Lepzig, 1956) and ^{40}K was measured by using a whole-body scintillation counter. Cardiac frequency (fh, 1·5) and ventilation ($\dot{V}_E$, 1·5) at an oxygen intake of 1·5 l/min were determined from the regression of fh and $\dot{V}_E$ on $\dot{V}_{O_2}$ respectively; the data for each individual being collected from at least five different discontinuous levels of work on a Müller bicycle ergometer (designed at the Max Planck Institute for Arbeitsphysiologie, supplied by Zentral Werkstatt, Gottingen). Expired air was collected for analysis into Douglas bags and ventilatory volume was monitored continuously with a low-resistance dry gas meter (Parkinson–Cowan Ltd) in conjunction with a rubber bellows to absorb peak flows (McKerrow, 1953) in a manner previously described (Davies & Shirling, 1968).

Cardiac and respiratory frequency were recorded continuously by using light-weight clip electrodes (Davies & Copeland, 1964) in conjunction with an ECG differential amplifier and linear instantaneous rate meter (Neilson, 1963) and a bead thermistor placed in the opening of the mouthpiece respectively.

Maximum oxygen uptake ($\dot{V}_{O_{2max.}}$) was measured in the upright position on the ergometer. The subjects breathed directly through an Otis–McKerrow low-resistance valve (diameter of the mouthpiece was 1 in) through a short length of 1 in internal diameter smooth-bore tubing into a wide-bore vinyl plastic Douglas bag which was suspended to decrease its resistance to flow. The total measured resistances to this system at flow rates of 100, 300 l/min were <1 cm and 2·5 cm of water respectively. Gas was tranferred directly into small (1·5 l) plastic sampling bags and analysed for O_2 and CO_2 content by using Servomex paramagnetic O_2 and Beckman infrared CO_2 analysers.

The analysers were calibrated at frequent intervals against the (Lloyd) Haldane Chemical method. The criterion for maximal load on the overall O_2-transporting system was that $\dot{V}_{O_2}$ should show no further rise with increasing rate of work, i.e. at least 2 $\dot{V}_{O_2}$ values at the plateau level must agree within $\pm 5\%$.

TABLE 2. Measurements of various respiratory, circulatory and anthropometric variables in eighty healthy male subjects grouped according to age (mean data); symbols are as in Table 1

Subjects	Lungs (ventilation)					Heart (circulation)							Tissues					Overall O_2 transport		
	Age (years)	FVC (l)	FEV (l)	$\dot{V}_{E}$, 1·5 (l/min)	$\dot{V}_{E_{max.}}$ (l/min)	Hb (g/100 ml)	THb (g)	BV (l)	HV (ml)	fh, 1·5 (beats/ min)	fh, 900 (beats/ min)	$fh_{max.}$ (beats/ min)	Ht (cm)	Wt (kg)	^{40}K (g)	TM (cm^2)	CM (cm^2)	$\dot{V}_{O_2max.}$ (l/min)	$LA_{max.}$ (mmol /l)	LA, 1·5 (mmol /l)
Group I																				
Mean	22·3	5·38	4·34	37·00	131·88	14·90	761	5·10	788	136	159	196	176	69·07	136	184	82	3·52	14·14	2·75
SD	±1·76	±0·77	±0·94	±4·44	±16·47	±1·40	±156	±1·07	±7·3	±13·5	±15·4	±5·9	±6·8	±8·76	±17·8	±97	±53	±0·40	±3·42	±0·79
n		22	22	22	22	22	19	19	21	22	22	22	22	22	22	21	21	22	22	16
Group II																				
Mean	27·8	5·21	4·33	40·03	139·82	14·75	740	5·02	910	135	156	191	174	76·52	142	197	91	3·31	14·65	3·29
SD	±1·35	±0·61	±0·48	±4·24	±19·14	±1·31	±76	±0·49	±11·8	±14·8	±11·3	±5·8	±6·9	±12·14	±16·1	±141	±59	±0·45	±3·30	±0·91
n		15	15	15	15	15	9	9	15	15	15	15	15	15	10	15	15	15	15	14
Group III																				
Mean	33·4	5·35	4·05	41·62	138·85	14·59	690	5·72	864	134	153	188	177	78·16	146	210	94	3·59	13·19	2·70
SD	±1·6	±0·85	±0·73	±6·24	±19·26	±1·13	±125	±0·97	±9·0	±17·3	±16·9	±4·8	±5·8	±9·23	±24·3	±134	±45	±0·56	±2·14	±1·10
n		12	12	12	12	12	7	7	12	12	12	12	12	12	8	12	12	12	12	10
Group IV																				
Mean	37·5	4·93	3·98	42·45	136·76	14·93	758	5·08	894	133	156	185	174	73·96	139	185	83	3·14	16·12	3·50
SD	±1·1	±0·77	±0·65	±4·99	±16·19	±1·27	±118	±0·68	±11·8	±15·9	±17·1	±6·4	±5·8	±8·13	±12·1	±101	±53	±0·28	±4·07	±1·01
n		11	11	10	10	11	8	8	9	11	11	10	11	11	11	11	11	10	11	10
Group V																				
Mean	42·9	5·12	4·26	45·13	133·13	14·36	757	5·26	890	131	155	184	179	81·81	136	205	86	3·10	13·76	4·00
SD	±1·5	±0·72	±0·68	±8·11	±24·32	±1·04	±118	±0·61	±8·7	±15·2	±16·5	±4·4	±7·0	±10·65	±23·9	±75	±64	±0·49	±1·29	±1·45
n		9	9	9	9	9	3	3	7	9	9	9	9	9	7	9	9	9	9	9
Group VI																				
Mean	47·2	4·35	3·77	43·84	133·09	14·90	—	—	989	133	156	179	176	79·19	144	181	90	3·06	14·92	4·31
SD	±8·8	±0·67	±0·64	±6·60	±16·01	±1·23	—	—	±13·8	±10·9	±13·3	±5·2	±6·8	±10·37	±21·1	±65	±81	±0·49	±6·11	±3·08
n		9	8	9	9	9	—	—	8	9	9	9	9	9	7	9	9	9	8	9

TABLE 3. Cross correlation matrix for indices showing correlation coefficients that are significant at or below 5% level for subjects below ($n = 49$) and above ($n = 29$) 35 years of age; symbols are as in Table 1

	> 35 years															
< 35 years	FVC	FEV	$\dot{V}_E$, 1·5	$\dot{V}_{E_{max.}}$	BV	THb	fh, 1·5	HV	Ht	Wt	^{40}K	TM	CM	$\dot{V}_{O_2max.}$	LA, 1·5	$LA_{max.}$
FVC	—	0·80	−0·52	...	0·54	...	...	...	0·37	...	...	...	...	0·37	...	...
FEV	0·68	—	−0·47	...	...	...	...	...	0·38	...	...	...	...	0·35	...	...
$\dot{V}_E$, 1·5	−0·37	−0·45	—	...	...	...	...	...	...	...	...	...	...	−0·35	...	...
$\dot{V}_{E_{max.}}$	...	0·38	...	—	...	...	−0·43	...	...	...	...	...	...	0·58	...	...
BV	0·39	0·39	...	0·57	—	0·85	...	...	...	...	...	...	...	...	...	...
THb	0·37	...	...	0·49	0·92	—	...	...	0·51	...	...	...	...	...	...	...
fh, 1·5	...	...	...	...	−0·38	−0·30	—	...	...	...	...	...	...	−0·40	...	...
HV	...	...	...	0·44	0·29	...	...	—	0·41	0·61	...	...	0·60	0·54	...	...
Ht	0·65	0·50	...	0·44	0·44	0·31	...	...	—	0·65	...	...	...	...	...	...
Wt	...	...	...	0·59	0·65	0·59	...	0·53	0·39	—	...	0·48	0·64	...	...	...
^{40}K	0·46	0·47	...	...	0·62	0·53	...	...	0·51	0·64	—	...	0·40	...	...	...
TM	...	...	...	0·53	0·67	0·56	...	0·45	...	0·75	0·58	—	...	...	...	...
CM	...	...	...	0·43	0·43	0·41	...	0·48	...	0·64	0·51	0·62	—	...	...	...
$\dot{V}_{O_2max.}$	0·38	0·36	−0·49	0·53	0·63	0·54	−0·43	0·35	0·50	0·53	0·59	0·53	0·56	—	0·43	−0·36
LA, 1·5	...	...	...	...	...	...	0·41	...	...	...	...	...	...	0·34	—	...
$LA_{max.}$	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	—

RESULTS

The mean values, range and the standard deviations of the principal indices are listed in Table 2. The overall linear correlation coefficients for eighty subjects in the study are significant at or below the 5% level and are listed in Table 3. For the purpose of later discussion they have been further subdivided under headings, under and over 35 years of age.

Ventilation

Dimensional factors. The FVC is closely associated with other lung dimensional and functional measurements, including $FEV_{1\cdot0}$ and $\dot{V}E$, 1·5 and significantly associated with $\dot{V}O_{2max.}$,

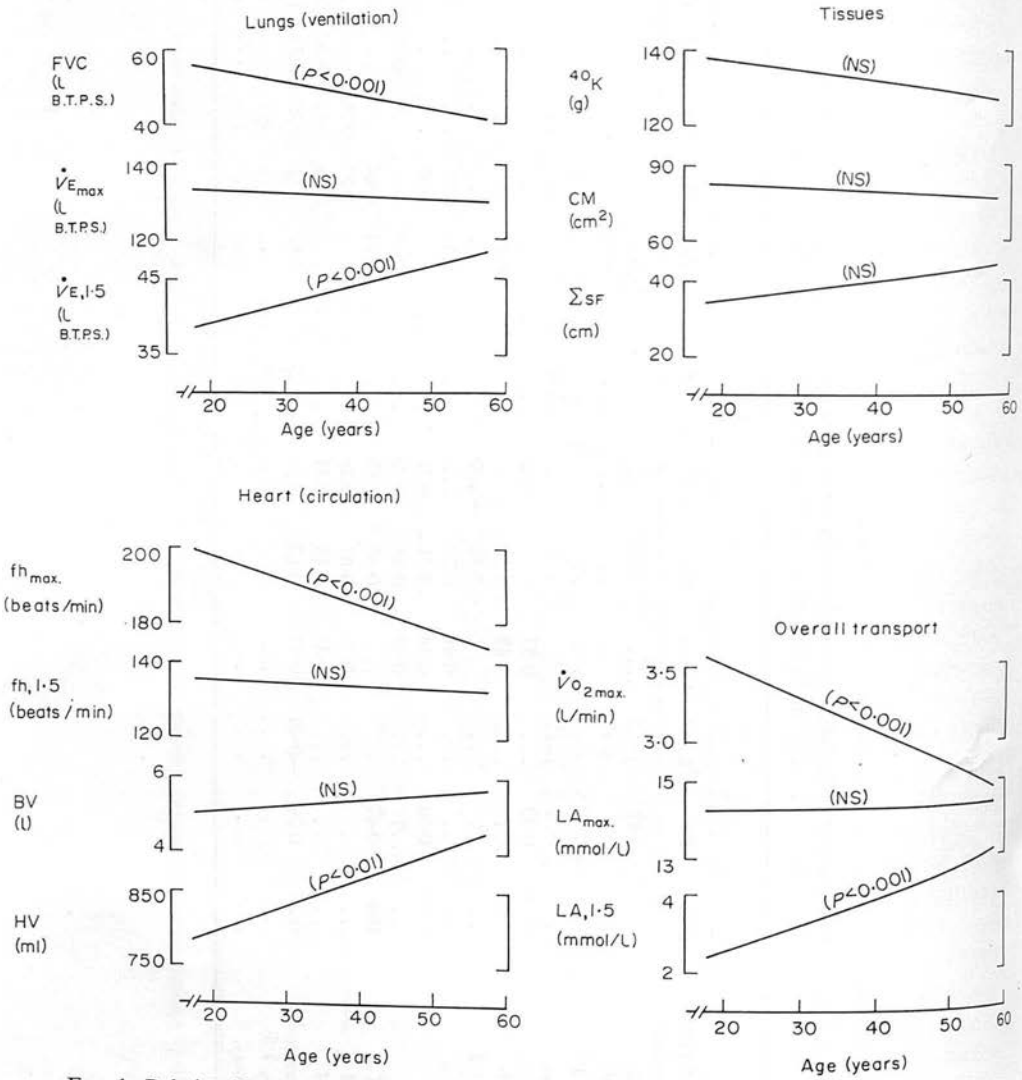

FIG. 1. Relationship of the functional and dimensional components of the O_2-transporting systems to age. Symbols are as in Table 1.

the overall correlation for the seventy-eight subjects being $r = 0{\cdot}44$. The absolute values of FVC decline with age (Fig. 1); the magnitude of this decrease together with average values for FVC are shown in Table 2. They are substantially in agreement with the values found by other workers (Berglund, Birath, Bjure, Grimby, Kjellmer, Sandquist & Soderholm, 1963; Kory, Callahan, Boren & Syner, 1961).

Functional factors. The ventilation at a given oxygen consumption of 1·5 l/min ($\dot{V}_E$, 1·5) shows an inverse relationship to $\dot{V}O_{2max.}$ but is unrelated to the maximum rate of ventilation ($\dot{V}_{E_{max.}}$, Table 3). This is true for all groups. The $\dot{V}_E$, 1·5 rises with age (Fig. 1) and since FVC falls it follows that the older subjects use a greater proportion of their expiratory reserve volume for a given $\dot{V}O_2$ than the younger subjects. The higher ventilation in subjects with a low $\dot{V}O_{2max.}$ is closely associated with the degree of anaerobiosis and the rise of blood lactic acid for given oxygen intake (LA 1·5) as shown in Fig. 1. The $\dot{V}_{E_{max.}}$ is positively related to $\dot{V}O_{2max.}$ and independent of age (Fig. 1). The average values for the six groups range from 131·9 to 139·8 l/min. These values are substantially higher than those reported by Robinson (1938) for subjects with similar range of aerobic power but the values for the young men (group I) are closely in agreement with those reported by Åstrand (1960).

When considering the association between the functional and dimensional components of any physiological system one cannot ignore the influences of common variables. Those most likely to affect the O_2-transporting system are, of course, body size and stature and the age of the individual subjects. To gain further information about this problem, partial regression equations of the various ventilation variables on age, weight and height were calculated. If weight or height or age or various combinations of these three variables were held constant, neither the correlations between $\dot{V}_E$, 1·5 and $\dot{V}O_{2max.}$ nor FVC and FEV were significantly changed. This was also true of the association between $\dot{V}_{E_{max.}}$ and $\dot{V}O_{2max.}$ However, the relationships of FVC and FEV to $\dot{V}O_{2max.}$ were decreased if height was held constant and disappeared when height and age were considered together.

Circulation

Dimensional factors. Blood volume (BV) and total haemoglobin are closely interrelated and both parameters are correlated with $\dot{V}O_{2max.}$ (Table 3). This relationship, however, is stronger in the younger age groups (Fig. 2, Table 3) and then gradually disappears with increasing age.

The average values for BV and THb range from 5·10 to 5·26 l and 690 to 761 g respectively. These values are lower than those reported by Holmgren & Åstrand (1966) for well-trained subjects and may thus be taken as more representative of sedentary subjects.

The BV is related to the size and stature of the subject, the overall correlation for the seventy-eight subjects with weight being 0·47 and height 0·51. However, if these variables are held constant a significant but decreased correlation with $\dot{V}O_{2max.}$ remains.

Heart volume is related to weight but not height. The overall correlation of HV with $\dot{V}O_{2max.}$ for seventy-eight subjects is poor (0·28) and not significant. However, this is partly due to the effect of age (Fig. 1). For each given age group listed in Table 2 a significant positive correlation exists ranging from 0·34 to 0·77. Heart volume is also inversely related to $fh_{max.}$ but with weight held constant this relationship disappears.

Functional components. The absolute value of the maximum cardiac frequency ($fh_{max.}$) for each group is given in Table 2. It can be seen that in the younger subjects fh_{max} reaches a value of 197 beats/min but thereafter declines with increasing age by approx. 5 beats/min per

decade. If weight is held constant the inverse relationship between $fh_{max.}$ and $\dot{V}O_{2max.}$ disappears, fh, 1·5 is negatively correlated with $\dot{V}O_{2max.}$ in the younger age groups but the relationship is decreased in the older subjects (Table 3). The overall correlation for the seventy-eight subjects with $\dot{V}O_{2max.}$ is −0·39. These relationships were unaffected by the height and weight of the subjects.

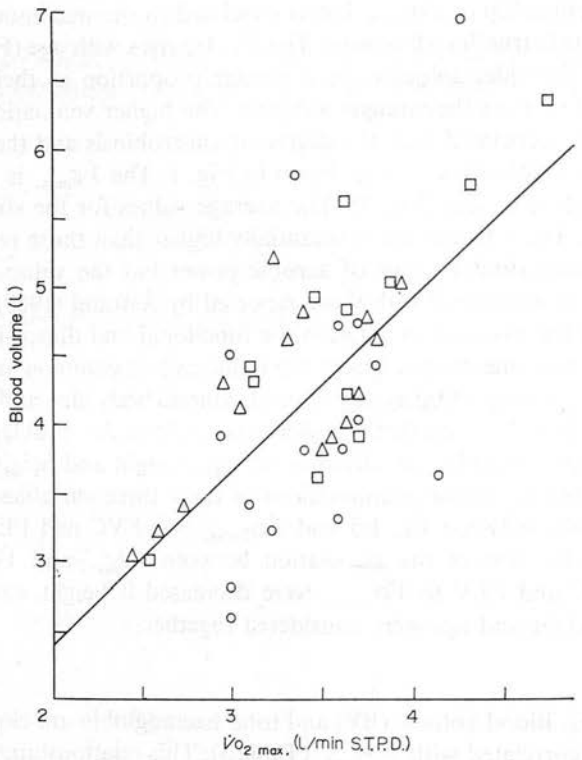

FIG. 2. Relationship between blood volume and maximum aerobic power ($\dot{V}O_{2\,max.}$) in younger subjects Groups I–III ($r = 0{\cdot}68$).

Tissues. For young subjects it will be seen that $\dot{V}O_{2max.}$ is highly correlated with body weight ($r = 0{\cdot}53$), height ($r = 0{\cdot}50$) and whole-body potassium ($r = 0{\cdot}59$) with estimates of thigh ($r = 0{\cdot}53$) and calf ($r = 0{\cdot}56$) muscle. The magnitude of the correlation coefficients and the average values shown in Table 2 are similar to those reported previously (Cotes *et al.*, 1969). However, in the older subjects the association between these variables completely disappears (Table 3) thus the overall correlation in the group as a whole is poor.

Components of the oxygen transporting system in relation to age. The relation of the ventilatory, circulatory and tissue components of the O_2-transporting system to age with body weight held constant is shown in Fig. 1. It will be seen that of all the various components measured only FVC and $fh_{max.}$ are associated with a decline of $\dot{V}O_{2max.}$. Heart volume and LA, 1·5 shows an increase whereas THb, fh, 1·5, estimates of thigh and calf muscle, ^{40}K, $\dot{V}E_{max.}$ and $LA_{max.}$ are largely independent of age from 20 to 50 years.

Prediction of maximum aerobic power. To investigate the predictive powers of the various parameters listed in Table 2 in relation to $\dot{V}O_{2max.}$ mutiple regression analyses were performed on the results. For subjects under 35 years of age the best single guide to $\dot{V}O_{2max.}$ was blood volume (BV). $\dot{V}O_{2max.}$ could be estimated with a coefficient of variation of 10%, from the following relationship:

$$\dot{V}O_{2max.} = 2{\cdot}12+0{\cdot}32\ BV.$$

In subjects over 35 years the best single guide to $\dot{V}O_{2max.}$ was given by HV from the formula:

$$\dot{V}O_{2max.} = 1{\cdot}29+0{\cdot}0022\ HV.$$

By using this equation maximum aerobic power could be estimated with a coefficient of variation of 12·2%.

When combining the two groups of subjects and considering the overall predictive powers of the various variables the best guide to $\dot{V}O_{2max.}$ was given by a linear regression on $\dot{V}E$, 1·5, fh, 1·5, and an estimate of calf (CM) and thigh (TM) muscle.

$\dot{V}O_{2max.} = 3{\cdot}69-0{\cdot}036\ \dot{V}E, 1{\cdot}5-0{\cdot}010$ fh, $1{\cdot}5+0{\cdot}13$ TM $+0{\cdot}12$ CM. (SD = 350 ml, coefficient of variation was 9%).

However, equally good prediction could be achieved from the regression on FVC, HV, and thigh muscle if age was included in the formula:

$\dot{V}O_{2max.} = 0{\cdot}03+0{\cdot}17$ FVC $+0{\cdot}0015$ HV $+0{\cdot}15$ TM $-0{\cdot}02$ Age (SD = 360 ml, coefficient of variation was 9%).

DISCUSSION

The results of this investigation were collected from normal healthy, but in the main, sedentary subjects. The values for maximum aerobic power are within the range given by Åstrand (1960) for a similar group of material and they therefore may be considered typical. Table 3 shows that the values for the various functional and dimensional components of the transporting system are well correlated to each other and to $\dot{V}O_{2max.}$, thus emphasizing the importance to body and organ size to power output in man. The mean values and the degree of intersubject variability are consistent in parts with the earlier data of Robinson (1938) and with those more recently reported (Holmgren & Åstrand, 1966). However, a few unexpected and important discrepancies do arise.

In the present study (Table 2 and Fig. 1) both $\dot{V}E_{max.}$ and $LA_{max.}$ are independent of age within the limited range of this study, i.e. 20–50 years. This contrasts markedly with the findings of Robinson (1938). He also gives values for $\dot{V}E_{max.}$ and $LA_{max.}$ respectively some 21% and 45% lower for his corresponding groups. It is unlikely that this discrepancy between the two studies arises solely from the selection of material, since in both sets of data the responses to submaximal exercise within each age group are almost identical and lung function values are within the normal range. The most likely explanation is that different criteria were used for evidence of maximal effort in the two investigations. In the study of Robinson (1938) the subjects were encouraged to run to exhaustion but no objective 'end point' was used. The subjects merely decided for themselves when they had reached the upper limits of performance. This procedure is obviously subjective and depends highly on motivation and the

degree of co-operation gained from the subject and undoubtedly leads to an under-estimation of maximum performance. In the present study the criterion for $\dot{V}O_{2max.}$ was that the $\dot{V}O_2$ showed no further rise with increasing work load, the plateau values of $\dot{V}O_2$ agreeing within $\pm 5\%$. This system was rigidly adhered to throughout the study and though fh and blood LA were used as guides to maximal effort they were never used as absolute criteria.

Both Åstrand (1952) and Holmgren & Åstrand (1966) give values for the dimensional components of the O_2-transporting system of young adults which are on average higher than those reported here. This is undoubtedly a reflection in part of the different work capacity of the two groups. Holmgren & Åstrand (1966) studied athletes and Åstrand (1952) investigated physical education students in whom the values for maximum aerobic power were well above the range given for this study. These workers also show very high correlation between function and dimensional capacities. Åstrand (1952) for instance gives a correlation of 0·96 between THb and $\dot{V}O_{2max.}$ and Holmgren & Åstrand (1966) give correlation coefficients ranging between 0·80 and 0·95 for nearly all the variables they studied, which is in marked contrast with the values given in Table 3 of the present investigation. Holmgren & Åstrand (1966) have admitted that their material was purposely highly selected and this must play a part in explaining the differences in association between dimensional and functional components within the investigations. However, it could also suggest that in the athlete and the well-trained subject the different components of the O_2-transporting system are closely integrated so that under conditions of load (i.e. during work) the size of each organ is more precisely matched to its functional capabilities, than in sedentary subjects. This also raises the possibility that different factors may set the upper limits of performance in the two groups, particularly when one takes age into account as in the present investigation.

Since the important dimensional and functional components of the O_2-transporting system have been shown either to increase or remain constant whereas $\dot{V}O_{2max.}$ decreases with age, it is expected that the prediction of maximum aerobic power from these variables without reference to age will be poor. Further when each variable is plotted against $\dot{V}O_{2max.}$ the scatter of individual points about the regression line suggests that the predictive power of a single variable is extremely low. This has already been shown for cardiac frequency (Davies, 1968). Measurements of fh and $\dot{V}O_2$ taken at four rates of work were found to give a crude estimate of $\dot{V}O_{2max.}$ coefficient of variation (12%) with a systematic under-estimation of $\sim 15\%$ due to the asymptotic nature of the fh/$\dot{V}O_2$ curve in untrained subjects.

On theoretical grounds the estimation of $\dot{V}O_{2max.}$ might be improved by the inclusion of additional variables. In younger and older subjects the best single guide to $\dot{V}O_{2max.}$ are BV and HV (with coefficients of variation of 10 and 12·2% respectively). If the two groups are considered together the best guide to $\dot{V}O_2$ is given by a multiple regression on either $\dot{V}_E$, 1·5, fh, 1·5 and leg muscle or FVC, HV, thigh muscle and age. These latter equations show a 2% improvement on conventional methods of predicting $\dot{V}O_{2max.}$ from measurements of fh and $\dot{V}O_2$ and the second equation had the additional advantage that it does not require the subject to exercise. It must be emphasized that the prediction of $\dot{V}O_{2max.}$ to within coefficient of variation of $\pm 9\%$ is still an extremely crude estimate of an individual maximum aerobic power. It means, for example, that if we have predicted $\dot{V}O_{2max.}$ of 3·4 l/min then we can say with 95% certainty that the true (observed) value will be somewhere between 2·8 and 4·0 l/min. Thus clearly in the individual case and particularly in all careful laboratory studies one must still encourage the direct measurement and reporting of $\dot{V}O_{2max.}$ However, in the

situation where large numbers of subjects are to be measured such as in community studies of PWC the situation is completely different. It is often unrealistic and impractical to make direct measurements of $\dot{V}O_{2max.}$ on a field-trial basis and one must decide in this situation whether an indirect and imprecise test can be substituted for a direct test of $\dot{V}O_{2max.}$ if one is only interested in differences between group means?

Table 4 shows the intra-subject variability of thirty-six measurements of $\dot{V}O_{2max.}$ made on six subjects. The average coefficient of variation is 3·5% (namely 1·9–6·2%), thus directly

TABLE 4. Intra-subject variation in subjects directly measured maximum oxygen intake ($\dot{V}O_{2max.}$) over a 6-day period

	$\dot{V}O_{2max.}$ (l/min)					
Day	Subject A	Subject B	Subject C	Subject D	Subject E	Subject F
1	3·44	3·13	2·97	3·21	3·15	3·01
2	3·30	2·97	2·79	3·42	3·36	2·79
3	3·45	3·07	2·98	3·32	3·36	2·92
4	3·48	3·11	2·94	3·48	3·48	3·00
5	3·56	3·18	3·32	3·49	3·57	3·10
6	3·36	3·08	3·25	3·43	3·44	3·23
Mean	3·43	3·09	3·05	3·39	3·39	3·08
SD	±0·08	±0·06	±0·19	±0·10	±0·12	±0·12
C.V.%	2·5	1·9	6·2	3·0	3·5	3·8

C.V. = Coefficient of variation

measured $\dot{V}O_{2max.}$ can be determined with an accuracy of ±110 ml. The inter-subject variability of $\dot{V}O_{2max.}$ for the present study is ±480 ml. By using these values together with those given above for the indirect estimation of $\dot{V}O_{2max.}$ by the multiple regression formulae, it is possible to calculate the number (n) of subjects required for any two populations to show a given difference between their means when using the direct or indirect test of $\dot{V}O_{2max.}$ as described by Pearson & Hartley (1966, Table 10, p. 141) for the estimation of population size. Applying such calculations to the present data they show that if we expect a 5% difference between the means $\dot{V}O_{2max.}$ of the populations, by using the direct test we must measure twenty subjects, and for the indirect test 100 subjects. If the difference is likely to be 10% then the values are 5 and 50 for direct and indirect respectively. Thus one can compensate for the loss of accuracy when using the simpler indirect test by increasing the number of subjects to be measured.

Finally one should stress that the prediction equations that were developed in a previous report (Cotes *et al.*, 1969) on young adults carried the implication that the lower $\dot{V}O_{2max.}$ of females compared with males was mainly due to their smaller organs including lungs, heart and muscle. This is still essentially true of the present results when one considers the relationship of the components of the transporting system to $\dot{V}O_{2max.}$ (Tables 2 and 3). In the younger subjects in particular the functional dimensional capacities of each organ system appear to be closely matched so as to bring forth on optimum transport of O_2 to the tissues. However, in the older subjects this hypothesis is no longer tenable. The size of organs (apart possibly

from the lungs, Fig. 1) for a given body weight appears to remain constant with increasing age whereas the $\dot{V}O_{2max.}$ declines. Thus it appears that an effect of age, particularly in sedentary subjects, is loss of the ability to maintain functional capabilities, despite retention of the dimensional capacity of youth. Åstrand, Engstrom, Eriksson, Karlsberg, Nylander, Saltin & Thoren (1963) have shown the same notable phenomena in young girl swimmers who were highly trained in their early teens for international swimming and who subsequently gave up the sport. On retesting 5 years after their last competitive event it was found that though dimensional capacities were retained their functional capabilities were decreased (Eriksson, Engstrom, Saltin & Åstrand, 1968). One may ask whether this decline in functional capacity is an obligatory and therefore necessary part of the ageing process or whether it is the culmination of events that could in part, be avoided. Holmgren (1967) has argued that the short-term effect of training is mainly regulatory leading to an increase in the functional aspects of the O_2-transporting system. Saltin and his co-workers (Hartley, Grimby, Kilbom, Nilsson, Åstrand, Bjure, Ekblom & Saltin, 1969; Saltin, Blomquist, Mitchell, Johnsson, Wildenthal & Chapman 1968) have shown that, whereas the increase in $\dot{V}O_{2max.}$ is small ($\sim$6·8%) on young healthy subjects, in man undergoing compulsory bed rest ($\sim$30%) or in middle-aged subjects it is much higher ($\sim$20%). If one applied the latter value to subjects in the 40–50 age range of the present study the decline of $\dot{V}O_{2max.}$ from 20 to 50 years would be considerably decreased if not completely abolished.

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Kinetics of cardiac output and respiratory gas exchange during exercise and recovery

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DAVIES, C. T. M., P. E. DI PRAMPERO, AND P. CERRETELLI. *Kinetics of cardiac output and respiratory gas exchange during exercise and recovery*. J. Appl. Physiol. 32(5): 618–625. 1972.—The kinetics of respiratory gas exchange and cardiac output were studied on a single-breath basis in two healthy male subjects, during onset and recovery, *a*) from rest to mild or *b*) to heavy exercise, and *c*) from mild to heavy exercise. Whereas in *a* and *b* the half-time of the process is about 30 s for $\dot{V}O_2$ and about 20 s for $\dot{Q}$, both at the onset of the exercise and in the recovery phase, in *c* the rate rise of $\dot{V}O_2$ is increased, the half-time being approximately 17–20 s, thus attaining the same value as for $\dot{Q}$. In the recovery from heavy to mild exercise the $\dot{V}O_2$ half-time change is of the order of 40 s. In the transition from rest to work (*a* and *b*) the cardiac output changes seem to be independent of $\dot{V}O_2$, which may in part reflect the influence of neurogenic control. The reduction of the $\dot{V}O_2$ half-time and therefore the similarity between $\dot{Q}$ and $\dot{V}O_2$ kinetics in the transition period from mild to heavier exercise may be explained on the basis of a prevailing metabolic drive upon $\dot{Q}$ mediated by an increased local vasodilation at the peripheral level.

unsteady state

THE KINETICS of the cardiac output ($\dot{Q}$) changes in the transition phase from rest to work, as calculated from the half-time ($t_{1/2}$), is of the order of 15 s, both in man and in the dog (2, 3). In man performing a submaximal exercise, the kinetics of the O_2 uptake adaptation to work is characterized by a $t_{1/2}$ of the order of 30 s. At the onset of work a difference seems therefore to exist between the $\dot{V}O_2$ and $\dot{Q}$ readjustments rates. This difference, however, is markedly reduced in the transition from light to heavier work, as in the latter case the $\dot{V}O_2$ half-time attains in man a value of 17 s (12). Correspondingly, the lactic acid (LA) formation which has been found to take place temporarily and to a limited extent in the transition from rest to work is, in the latter case, also reduced (12).

The aim of the present study is to examine the response of the heart minute volume ($\dot{Q}$) at the onset of activity, measured from different base lines of rest and mild steady exercise, in relation to the simultaneous $\dot{V}O_2$ changes.

PROCEDURE

The experiments have been made on two healthy subjects: *CTMD* (age, 34 yr; wt, 72 kg; $\dot{V}O_{2\,max}$, 3.4 l min^{-1}) and *PEdiP* (age, 28 yr; wt, 68 kg; $\dot{V}O_{2\,max}$, 3.3 l min^{-1}). They reported to the laboratory after a light breakfast or lunch and were repeatedly studied over a period of 8 weeks while performing each of the following work procedures: *a*) from rest to walking at 5.3 km/hr up a gradient of 5% and recovery, an exercise involving a net O_2 consumption of 18 ml/kg·min; *b*) from rest to walking at 7.8 km/hr up a gradient of 8%, involving a net O_2 consumption of 38 ml/kg·min; and *c*) during the transition from *a* to *b*. The combination of speed and gradient were chosen to correspond to ~40% and ~80% of the subjects' maximum aerobic power according to the data of Margaria (8).

Each subject upon arrival at the laboratory was allowed to rest, and during this preliminary period the electrodes for recording the ECG were fitted. After 20 min, the subject was asked to stand on the treadmill, and measurements of cardiac frequency (f_H), cardiac output ($\dot{Q}$), and respiratory gas exchange were made. The final 10 s were counted aloud, and at zero-time the subject stepped onto the treadmill belt, which had been previously set at the required speed and slope, and exercised for 10 min. Measurements of $\dot{Q}$, f_H, and respiratory exchange were then taken at frequent intervals throughout the work period. On cessation of exercise the subject stood astride the belt and measurements were continued for a 6-min period.

During the experiments of transition from light to heavier exercise or vice versa, the speed and slope of the treadmill were changed after 10 min of work and the subject continued at the new level of exercise for a further 10 min. The setting of the treadmill to the new work level was achieved within 10 s.

METHODS

Cardiac Output

Cardiac output was measured by means of modification of the indirect Fick technique first described by Kim, Rahn, and Farhi (7) from a record of the continuous breath by breath O_2 and CO_2 tension obtained from an Atlas mass spectrometer in conjunction with a Fleisch pneumotachograph and an *X-Y* recorder, as described by Cerretelli, Sikand, and Farhi (3).

Respiratory Gas Exchange

The expired air volume was obtained from the pneumotachograph tracing, and the O_2 consumption and CO_2 production obtained. The sampling line for the mass spectrometer was housed in a low-resistance valve (dead space, 50 ml) 3 cm from the lips of the subject.

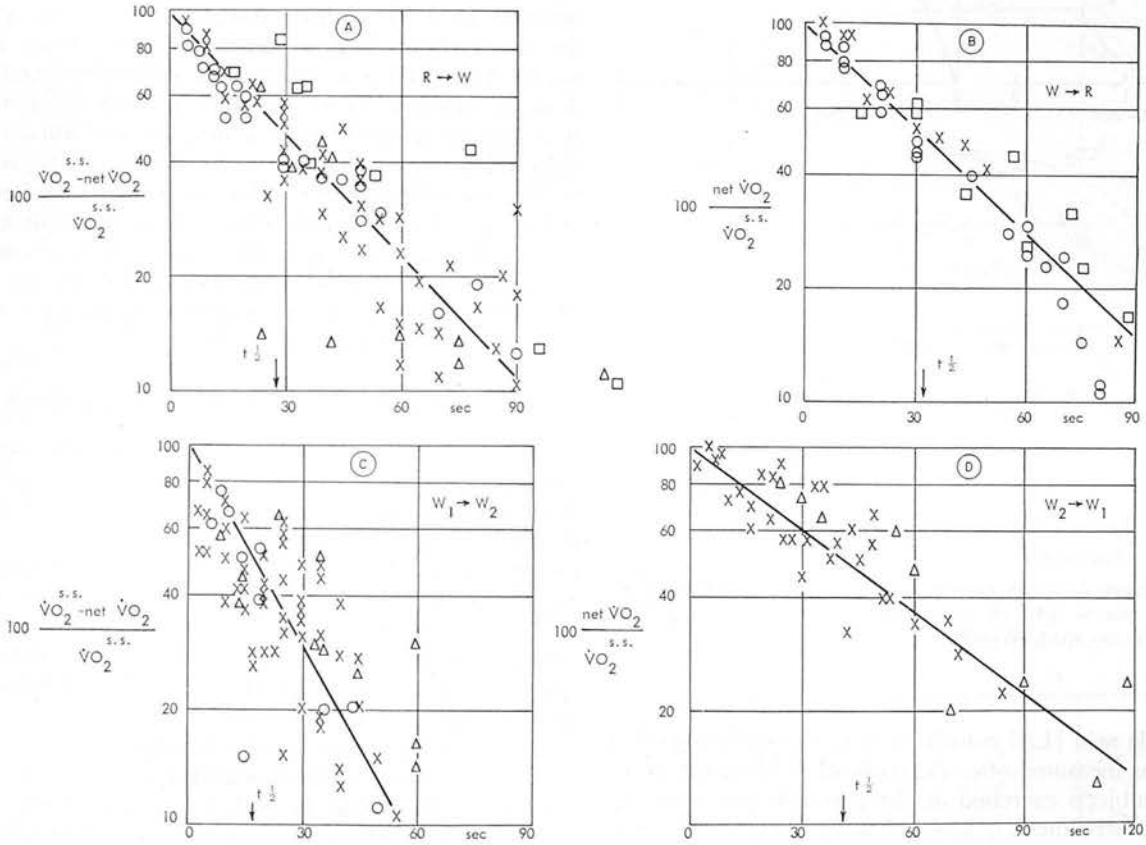

FIG. 1. Net O_2 intake as a function of time on a semilogarithmic scale. *A*, Rest to work (R→W); *B*, work to rest (W→R); *C*, light to heavy exercise ($W_1 \rightarrow W_2$), and *D*, heavy exercise to light exercise ($W_2 \rightarrow W_1$). Present data, △ light exercise (5.3 km hr^{-1} up 5% gradient) and □ heavy exercise (7.8 km hr^{-1} up 8% gradient), are superimposed on previous data for stepping and cycling collected in our laboratory (12). $\dot{V}o_2$ has been expressed as a percent of steady-state ($\dot{V}o_{2\,ss}$) condition.

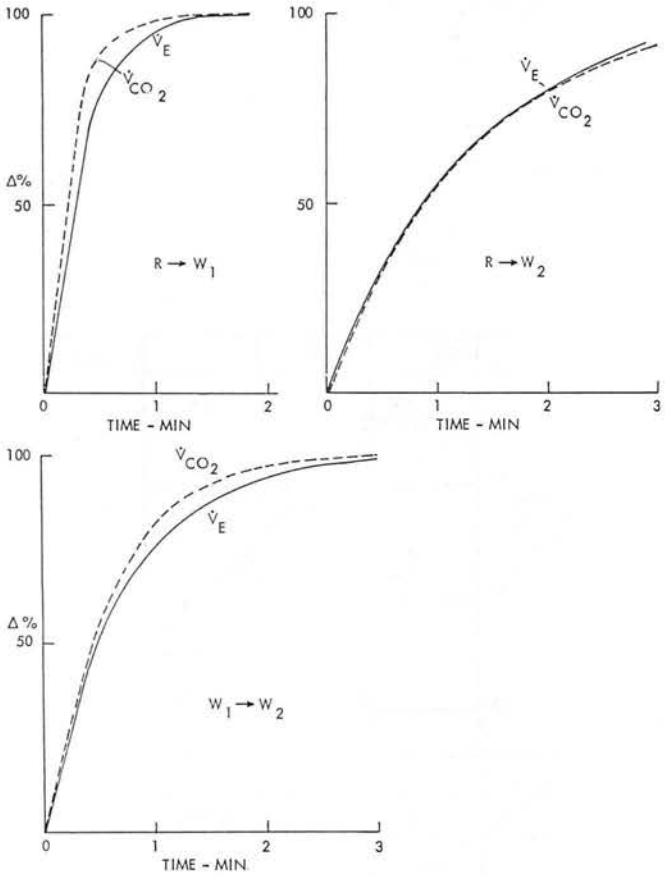

FIG. 2. Change ($\Delta\%$) in carbon dioxide ($\dot{V}_{CO_2}$) and ventilation ($\dot{V}_E$) during rest to light ($R \rightarrow W_1$) and heavy ($R \rightarrow W_2$) work and from light to heavy work ($W_1 \rightarrow W_2$).

Lactic Acid

The lactic acid (LA) contribution to metabolism during exercise was measured after the method of Margaria et al. (11): the subjects exercised at the two different work intensities for set times (1, 2, 4, 10 min, respectively), and blood was withdrawn from the cubital vein into 5-ml syringes at the 2nd and 4th min from the end of exercise. The determination of lactic acid was made by the enzymatic method of Gercken (5) and the maximal value attained in recovery was used in the calculation of the amount of LA produced by the muscles. This was obtained assuming that the distribution of LA in the whole body was such that the amount of LA in grams per kilogram of body weight was 0.75 the amount of LA in 1 liter of blood (10).

$$\text{g LA/kg} = 0.75 \text{ g LA/l}$$

RESULTS

Kinetics of Respiratory Gas Exchange During Exercise and Recovery

Oxygen intake. The data on O_2 intake during the transition from rest to work, and vice versa, and from light to heavier work and vice versa, are given in Fig. 1, together with data previously obtained on similar experiments (12). In all cases the kinetics of O_2 uptake appears to follow an approximately exponential function. It appears that: *a*) the half-time ($t_{1/2}$) of O_2 uptake in transition from rest to work or vice versa is about 30 s independent of the work load; *b*) in the transition from light to heavier exercise the rate of the O_2 uptake is higher ($t_{1/2}$ = 17 s); *c*) in the transition from heavier to lighter work it is lower ($t_{1/2}$ = 45 s).

Ventilation and carbon dioxide output. The changes of ventilation ($\dot{V}_E$) follow the same time course as the elimination of CO_2: since ventilation is merely controlled by the necessity to eliminate CO_2, it is not surprising to find these functions closely linked (Fig. 2). When proceeding from rest to light exercise the half-time of $\dot{V}_E$ or $\dot{V}_{CO_2}$ change is of the order of 15 s; when proceeding from rest to heavy exercise the $t_{1/2}$ is about 50 s, a value some 30% lower than the $t_{1/2}$ for $\dot{V}_{O_2}$. This is in agreement with earlier findings (2). A major outcome of the different kinetics of $\dot{V}_E$ and $\dot{V}_{CO_2}$ is a change in body CO_2 stores. In the transition from light to heavier work the process is faster, and $t_{1/2}$ is reduced to 30 s, the same relative reduction as in the rate of change of $\dot{V}_{O_2}$ (Fig. 1). During the change from heavy to light work the $\dot{V}_E$ declines at a very slow rate, the $t_{1/2}$ being of the order of 126 s. A slower kinetics of $\dot{V}_E$ during recovery from heavy compared with moderate work has been noted previously by Margaria et al. (11).

Kinetics of Circulatory Change in Exercise and Recovery

Cardiac frequency. At all levels of exercise the cardiac frequency (f_H) appears to anticipate $\dot{V}_{O_2}$. At the onset of work from rest the f_H rises at a very fast rate in both subjects. The steady-state f_H was reached within the 1st min for the mild exercise: at the higher level of exercise however, this initial rapid rise is followed by a more gradual increase in f_H (Fig. 3, *A* and *B*) even beyond the 3rd min. During the transition from light to heavy work this secondary gradual rise is not observed, the adjustment of f_H is practically complete within 1.5 min ($t_{1/2}$ = 22.5 s).

During the recovery period measured to a base line of rest, the f_H appears to lag behind the $\dot{V}_{O_2}$ changes. The half-time of the f_H change in both forms of recovery is ~70 s, compared with 30 and 45 s for $\dot{V}_{O_2}$, a value similar to that found by Margaria et al. (11) in recovery from heavy work.

Cardiac output. In the transition from rest to both light and heavy exercise the kinetics of cardiac output ()Q appears to anticipate the changes of $\dot{V}_{O_2}$ (Fig. 4); in the transition from light to heavier work, $\dot{Q}$ and $\dot{V}_{O_2}$ proceed at the same rate, the latter being faster than in the transition from rest to work. As in this case the heart rate follows the same time course as $\dot{V}_{O_2}$ and $\dot{Q}$, it follows that the stroke volume (SV) is substantially the same at both work levels (Fig. 3*B*).

Stroke volume. At rest SV is 78 ml; it rises to 120 ml at the lower and to 130 ml at the higher work level; the difference between the lower and the higher work load however is not statistically significant (Fig. 3*B*).

Blood Gas Changes in Exercise and Recovery

Venous CO_2 tensions. The time course of the mixed venous P_{CO_2} ($P\bar{v}_{CO_2}$) changes in exercise and recovery are shown in Fig. 5, *A* and *B*, in the transition from light to heavy work. In both figures a transient decrease in $P\bar{v}_{CO_2}$ takes place immediately upon the onset and cessation of work. These

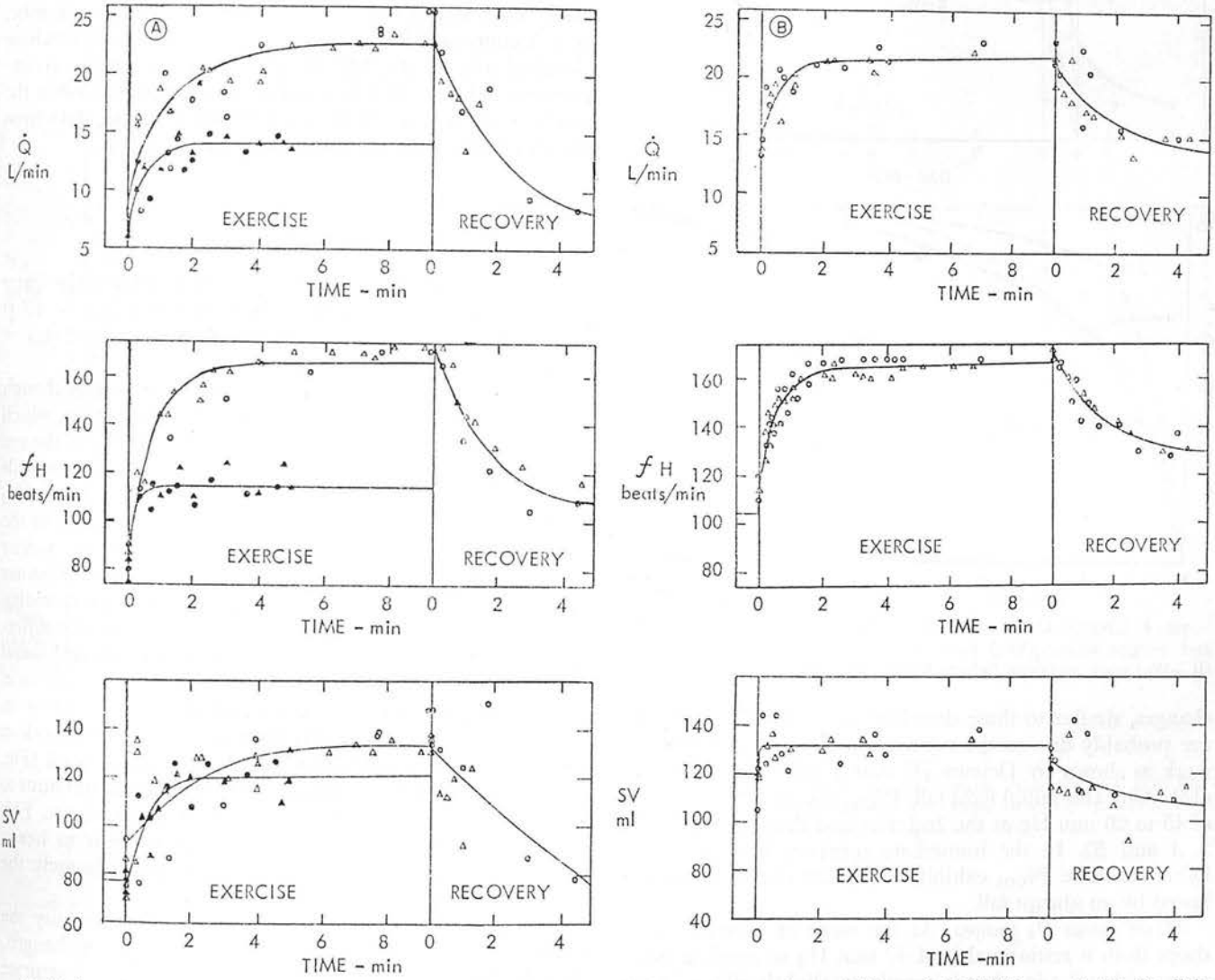

FIG. 3. Exercise and recovery curves measured from base lines of *A*) rest and *B*) mild steady exercise to (or from) different level(s) of work. From above downward: cardiac output ($\dot{Q}$), cardiac frequency (f_H), and stroke volume (SV). ▲ *Subj CTMD*, ● *subj PE diP*—light exercise (5.3 km hr^{-1} up 5% gradient); △ *subj CTMD*, ○ *PEdiP*—heavy exercise (7.8 km hr^{-1} up 8% gradient).

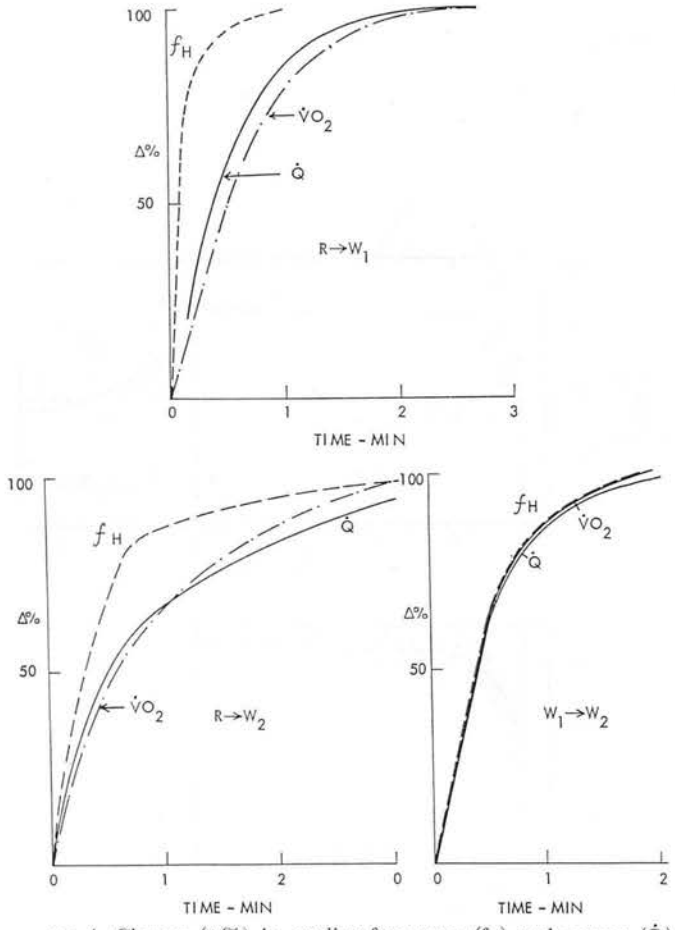

FIG. 4. Change (Δ%) in cardiac frequency (f_H) and output ($\dot{Q}$) and oxygen intake ($\dot{V}O_2$) from rest to light (R→W_1) and heavy (R→W_2) work and from light to heavy (W_1→W_2) exercise.

changes, similar to those described by Cerretelli et al. (3), are probably due to the neurogenic drive at the onset of work as shown by Dejours (4) and Torelli and D'Angelo (13). After this initial light fall, $P\bar{v}_{CO_2}$ rises from a rest value of 45 to 60 mm Hg at the 2nd min and then declines (Fig. 5, *A* and *B*). In the immediate recovery from heavy to lighter exercise $P\bar{v}_{CO_2}$ exhibits a definite transient rise followed by an abrupt fall.

Mixed venous O_2 tensions. At the onset of exercise $P\bar{v}_{CO_2}$ drops from a resting value of 40 mm Hg to reach a minimum within 2 min; then it overshoots slightly. $P\bar{v}_{CO_2}$ in the higher exercise is some 6 mm Hg lower than in lighter work; this difference is the same whether the heavy work was preceded by light exercise or not (Fig. 5*B*). During recovery from all three forms of exercise $P\bar{v}_{CO_2}$ recovers rapidly to its initial value with some evidence of a slight overshoot (Fig. 5, *A* and *B*).

Relation between $\dot{V}O_2$ and $\dot{Q}$; arteriovenous oxygen difference. In Fig. 6 O_2 uptake has been plotted as a function of cardiac output in the steady state (*A*) and in the transition phase from light to heavy exercise (*B*). It appears that $\dot{V}O_2$ is a linear function of Q in both cases; the isopleths indicating the arteriovenous O_2 differences which increases with $\dot{V}O_2$ but it does not differ significantly in the transition phase as compared with the steady-state condition. On the contrary, for a given $\dot{V}O_2$ value, Q appears to be in excess with reference to the steady-state work in the first 60–90 s during the transition from rest to work.

Lactic Acid Production

The lactic acid concentration in blood (Fig. 7) increases at the onset of exercise to reach a peak after about 3–6 min of the order of 7 mg/100 ml above basal for the light exercise and of 35 mg/100 ml for the heavier exercise. When the heavy work is reached from a condition of light exercise, the LA concentration increase is less pronounced, reaching a level of only 20 mg/100 ml. This indicates a reduced contribution of LA production to energy expenditure when the heavier work is reached from a previous exercise, than from rest, in accord with previous observations (12).

DISCUSSION

Kinetics of O_2 Debt Contraction

The kinetics of the O_2 uptake process appears to be faster in the transition from light to heavier work ($t_{1/2}$ = 17 s) than in the transition from rest to either work level ($t_{1/2}$ = 30 s).

It has been shown in a previous paper (12) that though the half-time of the gross alactic O_2 debt contraction, which includes the O_2 stores of the body, is 30 s, the $t_{1/2}$ of the net O_2 debt contraction is of the order of 17 s; this corresponds to an O_2 debt of 16 ml/kg for an exercise involving a $\dot{V}O_2$ of 40 ml/kg·min (as it is the case of the higher work in the present experiments). In the transition from rest to exercise the $t_{1/2}$ of the O_2 uptake process corrected for the O_2 stores (i.e., at the muscle level) amounts to 22 s (9), corresponding to an O_2 debt of 21 ml/kg for the same exercise; the difference (21 − 16 = 5 ml/kg) should represent an additional anaerobic energy source, which presumably is lactic acid production from glycogen. The lactic acid accumulation in blood is less in the transition from light to heavier work as compared with the transition from rest to heavy work (Fig. 7) and the difference expressed in O_2 equivalent[1] amounts to 12 − 7 = 5 ml/kg, the same order as given above. This indicates therefore that in transition from light to heavy exercise the O_2 uptake process follows approximately the same course as the net O_2 debt contraction.

During submaximal exercise whatever its intensity the amount of O_2 bound to myoglobin is probably unchanged, since the fall in O_2 in the muscle is not sufficient to desaturate myoglobin; the O_2 in venous blood also does not differ very appreciably between light and heavy work (Fig. 4*B*). The O_2 stores of the body therefore are not greatly different in these two conditions. This would indicate that in the transition from light to heavier work the actual change of O_2 stores of the body, in comparison with an exercise started from rest cannot influence appreciably the kinetics of $\dot{V}O_2$.

This supports the hypothesis that the gross alactic O_2 debt in submaximal exercise is made by *1*) the net O_2 debt, corresponding to the energy obtained from phosphagen breakdown, *2*) the oxidative energy drawn from the O_2 stores of the body, and *3*) by a limited amount of energy due to LA

[1] One gram per liter of increase of LA concentration in the blood is equivalent to an oxygen consumption of about 35 ml/kg (10) body wt.

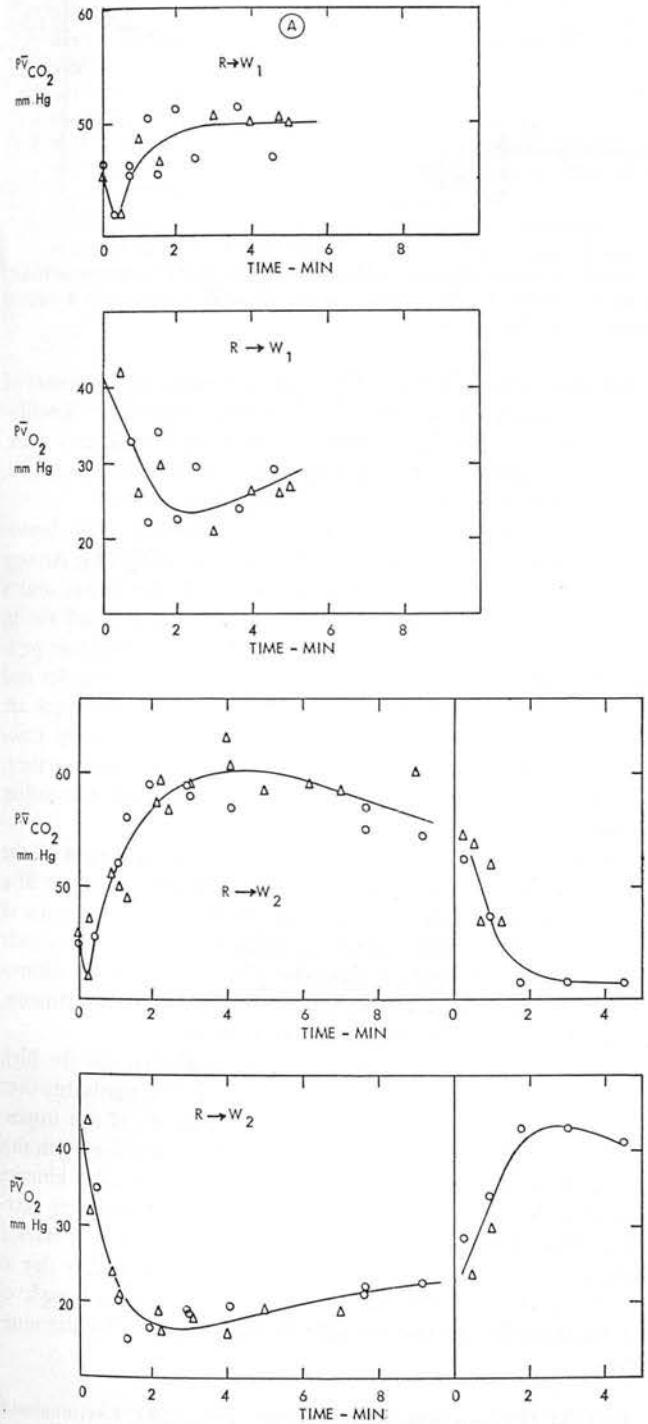

FIG. 5. Readjustments in mixed venous P_{CO_2} ($P\bar{v}_{CO_2}$) and P_{O_2} ($P\bar{v}_{O_2}$) gas tensions from *A*) rest to exercise ($R \rightarrow W_1$); $R \rightarrow W_2$), and *B*) from mild steady exercise to heavier work ($W_1 \rightarrow W_2$).

building from glycogen, a transient process at the onset of exercise.

The kinetics of $\dot{V}O_2$ in transition from heavy to lighter work is markedly slower than from either exercise level to rest. Similarly also cardiac output, cardiac frequency, and ventilation, and CO_2 output follow much longer time courses in transition from heavy to lighter work than in all other conditions. While these data seem to be statistically significant and based on a high number of observations, we cannot at this time suggest a valid interpretation of this phenomenon. The energy cost of the work of breathing and of the cardiac activity during the early phase of recovery may have some bearing on these findings.

Cardiac Output

In the transition from rest to work, $\dot{Q}$ seems to increase at a faster rate than $\dot{V}O_2$ independent of the work load, thus confirming previous observations (2, 3) and recent findings by Durand et al. (personal communication), who obtained a half-time value for the Q readjustments, at the onset of work from rest, of the order of 15–20 s, using the ^{85}Kr-dilution technique. In the transition from light to heavy work, the rate of the $\dot{V}O_2$ changes increases markedly, i.e., both $\dot{V}O_2$ and $\dot{Q}$ increases at the same rate, about 20 s.

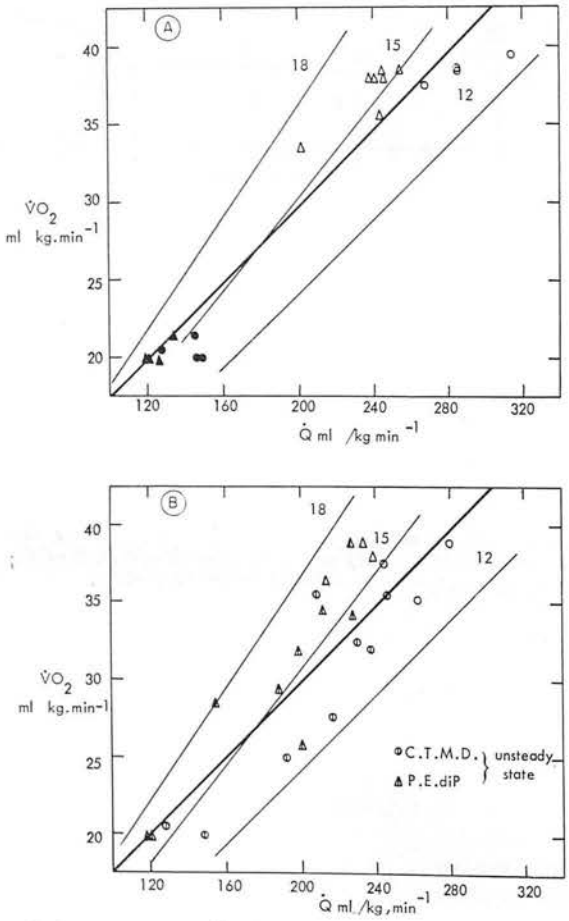

FIG. 6. Oxygen intake ($\dot{V}O_2$) and cardiac output ($\dot{Q}$) during the steady state of exercise *A*) rest to 5.3 km hr^{-1} up 5% gradient (symbols as for Fig. 3); and *B*) 5.3 km hr^{1}, 5%, to 7.8 km hr^{-1}, 8% gradient. Isopleths for arteriovenous difference are shown.

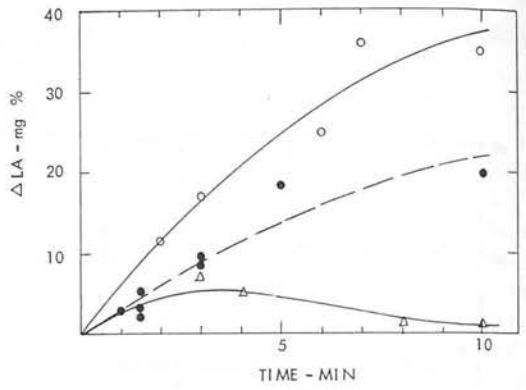

FIG. 7. Change of lactic acid concentration (ΔLA) in venous blood. Δ Rest to light (R$\rightarrow$W$_1$) and ○ heavy (R$_1\rightarrow$W$_2$) work and ● light to heavy (W$_1\rightarrow$W$_2$) exercise.

At the onset of exercise many neurogenic and hemodynamic factors contribute to the increase of $\dot{Q}$ (1). Among these are *a*) a sympathetic stimulation of the heart and a release of the vagal tone (1), both producing a rapid rise in heart rate; and *b*) a rapid increase of the mean systemic pressure caused by an instantaneous tensing of the muscles and by autonomic adjustments (6). These sudden changes are followed by an increase in muscle metabolism, local vasodilation, and a decreased resistance to venous return; together these factors provide a further stimulus to cardiac output.

The kinetics of the $\dot{Q}$ readjustment that is found in the various types of experiments are practically constant at a $t_{1/2}$ of about 20 s. This appears also to be the time course of the "true" O_2 consumption readjustments at the muscle level (6), thus suggesting that the kinetics of the simultaneous Q changes, as determined by the present experiments, is mainly dependent on metabolic events.

The pure neurogenic drive on $\dot{Q}$, as shown by the high kinetics of the heart rate change ($t_{1/2}$ = 7 s) is probably over in less than 10 s (6) and is therefore difficult, if not impossible, to detect by the method used for evaluating $\dot{Q}$ in this study. The observed difference in the O_2 uptake kinetics between the experiments in which the subject starts exercising from rest and those in which the exercise is started from a base line of mild exercise (Fig. 1) is probably due to the fact that in the latter case the O_2 uptake at the lung level is representative of the actual O_2 consumption by the muscles.

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Changes in mixed venous gas tensions at start of exercise in man

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EDWARDS, RICHARD H. T., DAVID M. DENISON, GERALD JONES, C. T. MERVYN DAVIES, AND E. J. MORAN CAMPBELL. *Changes in mixed venous gas tensions at start of exercise in man.* J. Appl. Physiol. 32(2): 165–169. 1972.—In three healthy men mixed venous P_{O_2} ($P\bar{v}_{O_2}$) and P_{CO_2} ($P\bar{v}_{CO_2}$) were estimated by a rebreathing technique before and after completing periods of upright exercise at 900 kpm min^{-1} (150 W) on a cycle ergometer. At rest $P\bar{v}_{O_2}$ and $P\bar{v}_{CO_2}$ averaged 35.5 ± 4.0 and 45.5 ± 2.5 (SD) mm Hg, respectively. $P\bar{v}_{O_2}$ fell after only 15 s of work and reached a minimum of 22.5 ± 1.0 mm Hg at 1 min, suggesting that the circulatory adaptation lagged behind the rising tissue oxygen consumption. $P\bar{v}_{CO_2}$ started to rise with a 30-s delay from the start of exercise, probably because of alveolar overventilation, and reached a maximum of 61.5 ± 3.1 mm Hg after 5 min. Experimental hypocapnia, hypercapnia, hypoxia, and hyperoxia in the 1st min of exercise greatly altered end-tidal P_{O_2}, P_{CO_2}, ventilation and $P\bar{v}_{CO_2}$ but had less influence on heart rate and $P\bar{v}_{O_2}$. These respiratory stresses were without influence on exercise performance, and their effect on the measured variables was short-lived, suggesting that there exists a reserve capacity in the homeostatic mechanisms at the start of work.

ventilation; circulation; hypocapnia; hypercapnia; hypoxia; hyperoxia; heart rate

FOR MANY YEARS rebreathing techniques have been used in an attempt to estimate the mixed venous gas tensions in man. The early workers (2, 3, 16, 25) were interested in the measurement of cardiac output. Since this measurement was subsequently made with greater precision and convenience by other methods the rebreathing techniques were largely forgotten. They were subsequently reintroduced, with the aid of mass spectrometry, for estimating cardiac output (4, 11) and tissue gas tensions (12, 20) noninvasively in man. Hitherto measurements of the mixed venous P_{CO_2} ($P\bar{v}_{CO_2}$) and P_{O_2} ($P\bar{v}_{O_2}$) have been made in relatively steady states at rest, during exercise, and when a variety of respiratory stresses have been experimentally imposed (12).

This paper describes the changes in mixed venous gas tensions during unsteady states, at the start of moderately severe upright exercise when adaptations were allowed to proceed naturally and when experimental hypocapnia, hypercapnia, hypoxia, or hyperoxia were imposed during the 1st min of exercise. Some of the results have been reported in a preliminary form (17).

METHODS

Anthropometric details of the three healthy men studied are given in Table 1. The subjects exercised wearing shorts and shirt in a laboratory in which the temperature was within the range 19–24 C during the series of experiments described. Subjects exercised in the upright position on an electrically braked cycle ergometer (Elema). The power output (900 kpm min^{-1}, 150 W) represented about 70% of the subjects' maximal oxygen intake estimated from the heart rate at several submaximal work levels (8).

Resting measurements of heart rate, ventilation, and end-tidal and mixed venous gas tensions were made while the subject was seated on the cycle ergometer. The subject then abruptly started to exercise, achieving the standard power output in less than 2 s. Each work period was terminated by an estimation of the mixed venous gas tensions by rebreathing (below). The time course of changes in mixed venous gas tensions was determined by altering the duration of exercise before rebreathing. Runs of 15 s to 3 min duration were repeated in reverse order to minimize the effects of repeated exercise (9). The time course of changes in end-tidal gas tensions, ventilation and heart rate were constructed from the results of 10 runs of 5 min duration on each subject. The sequence of runs was designed in such a way that the subject exercised regularly for about 5 min every 30 min.

Two types of study were performed: *1*) of changes at the start of exercise when adaptations were allowed to proceed normally, and *2*) of the effects of preventing or exaggerating the usual changes in pulmonary O_2 and CO_2 exchange during the early phase of exercise by imposing the following respiratory stresses for the 1st min: *a*) hypocapnia—subjects hyperventilated voluntarily from the start at a level approximately equal to that in the 5th min of exercise (18); *b*) hypercapnia—subjects rebreathed from a 6-L bag containing 7–9% CO_2 in oxygen; *c*) hypoxia—subjects breathed a gas mixture containing 10.7% O_2 in N_2; *d*) hyperoxia—subjects breathed pure oxygen.

In the second study the time course of the mixed venous gas tensions was constructed from measurements during runs of 1, 2, and 5 min duration.

A respiratory mass spectrometer (AEI MS 4) was used to measure the partial pressures of O_2, CO_2, and N_2 in the expired air sampled from the axial stream 3 cm from the lips. Signals corresponding to the pressure of each of these gases were recorded on separate channels of a Sanborn four-

TABLE 1. *Anthropometric data on the subjects*

Subj	Age, yr	Ht, cm	Wt, kg	FEV_1, L BTPS	VC, L BTPS	T_{LCO}, ml mm Hg^{-1}	Hemoglobin, g 100 ml^{-1}	Estimated $\dot{V}_{O_2max}$, L STPD min^{-1}
1	29	178	75.5	4.1	4.7	34	14.8	3.3
2	35	179	70.0	5.2	6.1	40	15.9	3.5
3	35	179	73.2	4.0	4.8	45.5	13.7	3.4

FEV_1 = forced expired volume in 1 s; VC = vital capacity; T_{LCO} = transfer factor for carbon monoxide.

channel recorder. The mass spectrometer was used at its maximum sensitivity of 0.7–1.0 mm Hg/mm paper for each of the gases.

The mixed venous gas tensions were estimated by the following technique (4, 11). Subjects expired to residual volume and rebreathed a CO_2 in N_2 mixture at a rate of 1 breath/s for 8–10 breaths. Simultaneous recording of O_2, CO_2, and N_2 pressures in the respired gas throughout the period of rebreathing allowed the recognition of a mixing phase lasting 2–3 s followed by an equilibration phase (plateau) lasting until the tenth second after which there was a gradual change due to recirculation. Proper adjustment of the volume and carbon dioxide concentration of the initial mixture (18) enabled us to obtain a nearly horizontal plateau in almost all cases. The plateau was extrapolated to 30 s at rest and 20 s on exercise as the extrapolated value was known to lie within 1 mm Hg of the true plateau value (11, 18) and compared favourably with that in directly sampled mixed venous blood (13).

Estimates of the $P\bar{v}_{CO_2}$ in the hypercapnia and hyperoxia experiments were corrected for the high alveolar oxygen tension (Haldane correction, 12) to make the values comparable with the true mixed venous tensions determined in *study 1* and the hypocapnia and hypoxia runs of *study 2*.

Expired ventilation was measured with a bellows spirometer mounted directly above the respiratory valve and mouthpiece assembly. The spirometer emptied by gravity through an electropneumatic exit valve which was triggered by the onset of inspiratory gas flow. A maximal expiration into the spirometer against a closed exit valve resulted in a static pressure of 3 cm H_2O. The spirometer had a linear calibration for respiratory frequencies up to 35 breaths/min, a figure not exceeded in any of the studies. Heart rate was measured continuously with a cardiotachometer (Devices).

The electrical signals from the potentiometer on the spirometer, ECG amplifier, and cardiotachometer were recorded with a direct-writing oscillograph (Minograf 81).

RESULTS

At rest the mean values (±SD) for the mixed gas tensions were: $P\bar{v}_{CO_2}$ = 45.5 ± 2.5 mm Hg, $P\bar{v}_{O_2}$ = 35.5 ± 4.0 mm Hg (n = 42). The changes in mixed venous gas tensions at the start of exercise (*study 1*) are shown in Fig. 1. A significant fall ($P < 0.01$) in $P\bar{v}_{O_2}$ occurred in the shortest exercise runs of 15 s duration, $P\bar{v}_{CO_2}$ did not increase until exercise had been sustained for more than 30 s. After 5 min of exercise the mixed venous gas tensions averaged: $P\bar{v}_{O_2}$ = 25.5 ± 1.3 mm Hg (n = 30) and $P\bar{v}_{CO_2}$ = 61.5 ± 3.1 mm Hg (n = 30). At rest the end tidal gas tensions were 33.5 ± 5.0 mm Hg (n = 33) for P_{CO_2} and 108.5 ± 9.0 mm Hg (n = 30) for P_{O_2}.

The end-tidal gas tensions immediately prior to rebreathing in runs of 15 s to 3 min duration were close to those found at comparable times after the start of exercise in the 5-min runs (Fig. 2).

Ventilation and heart rate increased approximately exponentially during the 5 min of work (Fig. 3) from the immediate preexercise values of 10.4 ± 2.0 L BTPS min^{-1} (n = 24) and 100 ± 10 beats min^{-1} (n = 26) to 56.9 ± 5.2 L BTPS min^{-1} (n = 21) and 170 ± 4 beats min^{-1} (n = 25), respectively. The experimental alterations in pulmonary gas exchange during the 1st min of exercise were well tolerated by the subjects.

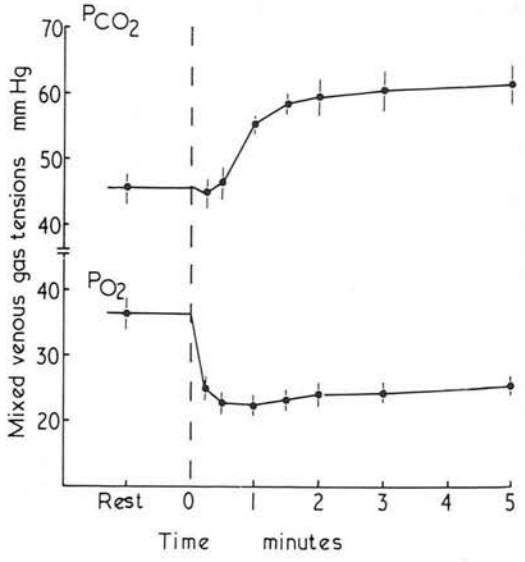

FIG. 1. Changes in mixed venous gas tensions during the first 5 min of exercise (mean ± 1 SD shown, n = 6 for all except rest and 5-min values when n = 42 and n = 30, respectively).

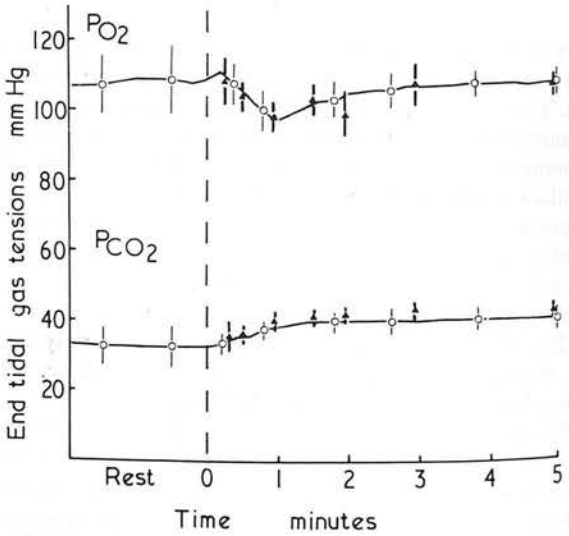

FIG. 2. Changes in end-tidal gas tensions during the first 5 min of exercise (mean ± 1 SD shown, n = 11). Solid triangles are measurements made immediately prior to rebreathing in runs in which mixed venous gas tensions were measured (mean ± 1 SD shown, n = 6).

The effects on mixed venous gas tensions of hypocapnia, hypercapnia, hypoxia, or hyperoxia (*study 2*) are shown in Fig. 4. The time course of the changes in mixed venous gas tensions associated with the normal adaptation to exercise (Fig. 1) are superimposed in Fig. 4 for comparison. After 1 min of exercise with hypocapnia or hypoxia the mixed venous P_{CO_2} was significantly reduced ($P < 0.01$) by 8 and 6.5 mm Hg, respectively. Hypercapnia and hyperoxia caused significant increases ($P < 0.01$) in $P\bar{v}_{CO_2}$ of 11.5 and 10 mm Hg over the same period of exercise. At 1 min $P\bar{v}_{O_2}$ was unaffected by hypocapnia but was 5 mm Hg lower ($P < 0.01$) in the hypoxia runs. After exercise for 2 min, as after 5 min, mixed venous gas tensions were closely similar

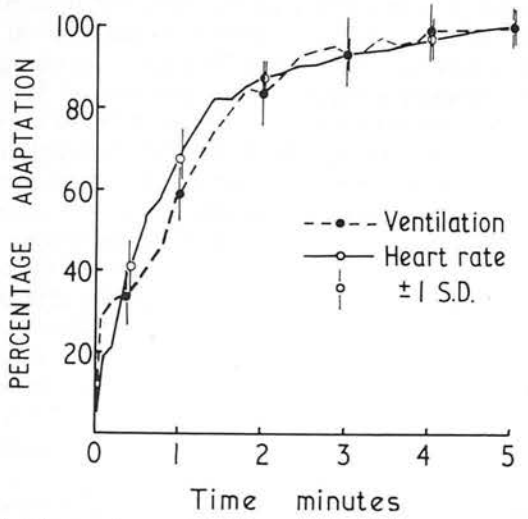

FIG. 3. Changes in ventilation and heart rate at the start of exercise (mean values ± 1 SD, n = 24, shown as a percentage of the average change at 5 min).

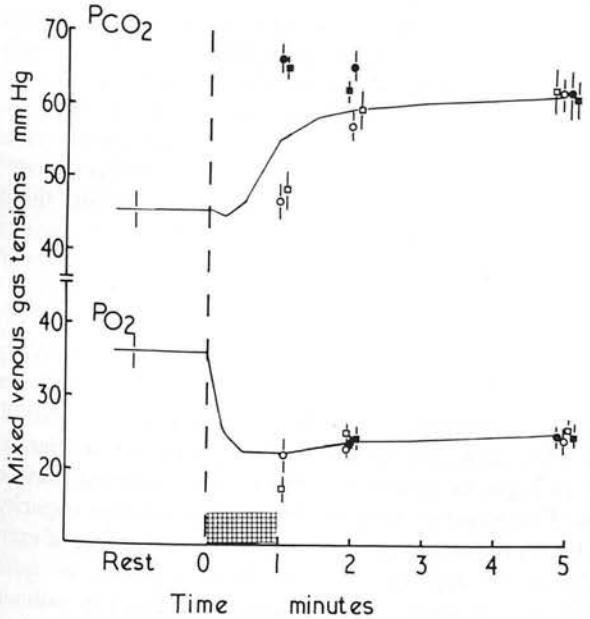

FIG. 4. Effects on mixed venous gas tensions of altering normal gas exchange in the first minute of exercise (shaded area). Open circles—hypocapnia, closed circles—hypercapnia, open squares—hypoxia, closed squares—hyperoxia; mean values ± 1 SD (n = 6) shown with normal pattern of changes given in Fig. 1 superimposed.

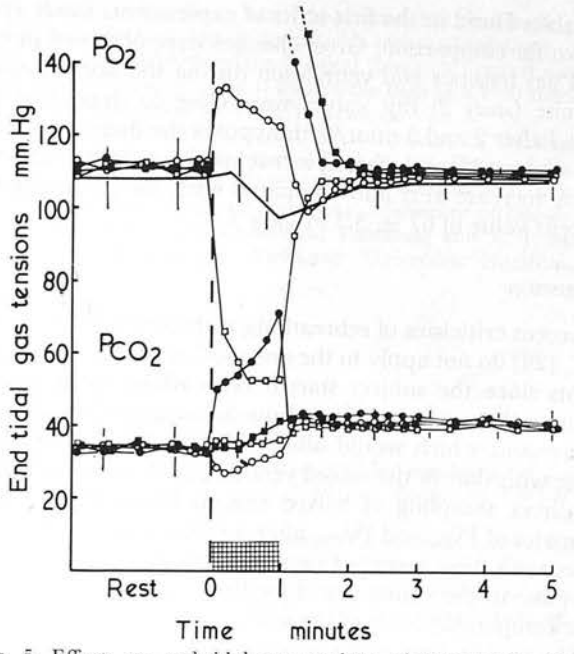

FIG. 5. Effects on end-tidal gas tensions of hypocapnia, hypercapnia, hypoxia, or hyperoxia during 1st min of exercise. Symbols as in Fig. 4. Normal pattern of changes at the start of exercise given in Fig. 2, is also shown.

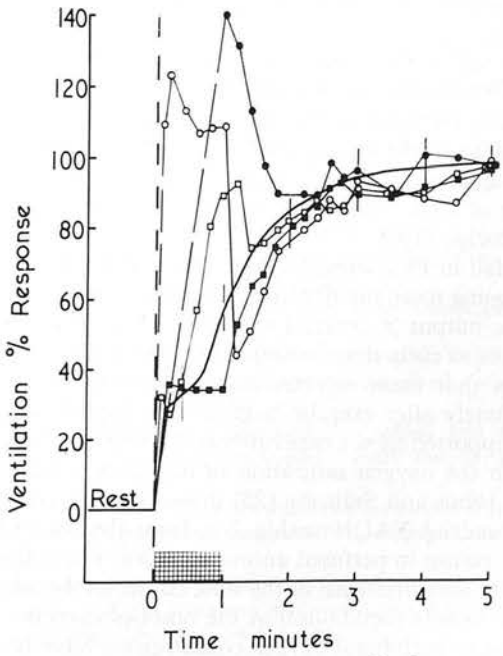

FIG. 6. Effects on ventilation of hypocapnia, hypercapnia, hypoxia, and hyperoxia during 1st min of exercise. Normal pattern of adaption given in Fig. 3 is superimposed.

in the two series of experiments (*studies 1* and *2*). It was not possible to determine $P\bar{v}_{O_2}$ at 1 min in the hypercapnia and hyperoxia runs owing to the high alveolar oxygen pressure prior to rebreathing. Previous measurements during steady-state exercise (12) suggest that values would have been close to 40 mm Hg at 1 min in these runs.

In the figures showing the influence of hypocapnia, hypercapnia, hypoxia, and hyperoxia on end-tidal gas tensions (Fig. 5) and ventilation (Fig. 6) the time course of these

variables found in the first series of experiments (*study 1*) are shown for comparison. Gross changes were observed in end-tidal gas tensions and ventilation during the first minute of exercise (*study 2*) but values were close to those found in *study 1* after 2 and 5 min. With hypoxia the increase of heart rate was significantly faster, it reached 91 ± 11.2% at 1 min of the increase at 5 min compared with the corresponding percent value of 67 ± 3.3 in *study 1*.

DISCUSSION

Recent criticisms of rebreathing techniques for measuring $P\bar{v}_{O_2}$ (28) do not apply to the one used in the present experiments since the subject started rebreathing from residual volume, thus reducing the volume of oxygen in the lungs to an amount which would allow the alveolar P_{O_2} to equilibrate with that in the mixed venous blood, as demonstrated by direct sampling of mixed venous blood (13). Present estimates of $P\bar{v}_{O_2}$ and $P\bar{v}_{CO_2}$ after 5 min of exercise generally agree with those reported previously (4, 6, 12, 13) and they are close to the values found for femoral venous blood (14) at a comparable level of exercise.

The new findings of the present studies are the early fall in $P\bar{v}_{O_2}$ and the delayed rise in $P\bar{v}_{CO_2}$ (*study 1*), and the effects of hypocapnia, hypercapnia, hypoxia, or hyperoxia during the 1st min of exercise on mixed venous gas tensions, heart rate, pulmonary ventilation, and end-tidal gas tensions (*study 2*).

Early fall in $P\bar{v}_{O_2}$ (*study 1*). There has been no relevant study with the results of which this aspect of our study can be directly compared. $P\bar{v}_{O_2}$ fell more rapidly in our experiments than might be expected from the time taken for the oxygen content of femoral venous blood to fall at the beginning of mild supine exercise in patients with rheumatic heart disease (15).

The fall in $P\bar{v}_{O_2}$ suggests that tissue oxygen consumption is increasing more rapidly than circulatory oxygen transport (cardiac output × arterial oxygen content) (12, 29). That $P\bar{v}_{O_2}$ falls so early despite finite circulatory transit times (1) suggests that tissue oxygen consumption increases almost immediately after exercise begins. This hypothesis appears to be supported by the experiments of Millikan (24) showing a fall in the oxygen saturation of myoglobin within 0.2 s, and of Jöbsis and Stainsby (23) demonstrating oxidation of mitochondrial NADH within 5 s, from the start of a sustained tetanus in perfused animal muscles. There have been no direct measurements of the time course of the increase in aerobic muscle metabolism at the onset of exercise in man. Changes in peripheral oxygen consumption have been computed from measurements of cardiac output and alveolar oxygen transfer in man by Gilbert, Auchincloss, and Baule (22). These authors reported a faster increase in peripheral oxygen consumption than of cardiac output during the transition from rest to exercise. The part played by myoglobin as an oxygen store is difficult to assess but its effect in delaying the fall in $P\bar{v}_{O_2}$ is likely to be only transient at the present work level (19, 21).

Two other factors may be contributing to the early fall in $P\bar{v}_{O_2}$.

1) A sudden surge of blood with a low oxygen saturation from stagnant pools in the legs due to activation of the "muscle pump" at the onset of work. We have measured neither the oxygen content nor the volume of blood pooled in the legs of our subjects prior to exercise. The oxygen content is unlikely to be less than 4%, the lowest content found in the femoral venous blood of subjects standing on a treadmill immediately before exercise (26). Nearly one-third of the total blood volume, consisting of blood with this oxygen content, would have to be admixed with resting mixed venous blood to account for the observed fall in $P\bar{v}_{O_2}$; an even larger volume would be required were the oxygen content higher. Other authors (22, 27) estimate that no more than 15% of the total blood volume is pooled in the legs in the upright posture so that admixture of blood is likely to be contributing only a fraction of the observed fall in $P\bar{v}_{O_2}$.

2) A fall in arterial P_{O_2}. In the studies of Bjurstedt and Wigertz (1) the arterial P_{O_2} started to fall after 30 s and reached a minimum of 15 mm Hg below the resting level 90 s after the start of supine leg exercise in man. The fall in arterial P_{O_2} was attributed by these authors to an imbalance between the O_2 uptake from, and O_2 supply to, the alveolar gas since ventilation increased more slowly than circulation (heart rate) in response to a step increase in power output (loadless pedaling → 650 kpm min^{-1}). In the present experiments the time course for the increase in ventilation closely followed that for heart rate (Fig. 3), and there was no significant fall in end-tidal P_{O_2} (Fig. 2). These facts suggest that the initial rates of change of ventilation and circulation were fairly well matched in our subjects. However, even if a fall in arterial P_{O_2} had occurred it would have been too late to contribute appreciably to the early fall in $P\bar{v}_{O_2}$ observed.

Delayed rise in $P\bar{v}_{CO_2}$ (*study 1*). This finding suggests that alveolar ventilation was relatively high for the amount of CO_2 being produced during the first 30 s of work (12, 29). The sharp "neurogenic" (10) rise in ventilation observed (Fig. 3) provided an initial acceleration of alveolar ventilation: the time course of the increase in tissue CO_2 production is more difficult to estimate. When, and how much, increased metabolism in exercising muscle influences the composition of mixed venous blood is determined by the circulatory transit time and the immediate storage capacity for CO_2 of the exercising muscle and venous blood. The early fall in $P\bar{v}_{O_2}$ makes it impossible to attribute the delay in the rise of $P\bar{v}_{CO_2}$ entirely to the circulatory transit time between exercising muscle and lung; the possibility of an alteration in CO_2 storage capacity must be considered. The immediate storage capacity of the whole body for CO_2 is greater in the steady state of exercise than at rest (5), but it is not known how soon after the start of exercise CO_2 storage capacity increases. It was shown (18), however, that blocking CO_2 excretion by rebreathing, did not accelerate the rise in $P\bar{v}_{CO_2}$ for more than 20 s after the onset of heavy exercise. This suggests that the immediate storage capacity for CO_2 can be instantly enlarged at the beginning of exercise.

Effects of hypocapnia, hypercapnia, hypoxia, or hyperoxia (*study 2*). All these experimental alterations in pulmonary gas exchange during the first minute of exercise resulted in gross changes in end tidal gas tensions (Fig. 5) and ventilation (Fig. 6) but hypoxia alone influenced heart rate. Hypoxia and hypercapnia exaggerated chemical drives to

ventilation (7). Hyperoxia depressed ventilation though the neurogenic rise in ventilation remained essentially the same as in the air breathing runs of *study 1*. The changes in mixed venous gas tensions were similar to those found previously with comparable respiratory stresses during steady state sub-maximal exercise (12). The respiratory stresses studied were well tolerated and all the measured variables promptly returned to values similar to those found in *study 1* when the stresses were removed. This suggests that there exists a large reserve capacity in the homeostatic mechanisms operating at the start of exercise.

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Plasma FFA in Relation to Maximum Power Output in Man

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Summary. The relation of plasma FFA to Lactic acid (LA) concentration and maximum aerobic power ($\dot{V}_{O_2}$max) was studied on 7 healthy, male subjects, ages 21—41 years, during work on a motor driven treadmill. The results showed (in agreement with the work of Issekutz *et al.* [19] on dogs) that there is a reciprocal relationship between plasma FFA and LA concentration and both are dependent on an individual's $\dot{V}_{O_2}$max. Below exercise levels corresponding to 65% $\dot{V}_{O_2}$max, FFA and LA remain fairly constant but beyond this critical level FFA falls precipitously and a marked rise in LA occurs. Calculation of the "Aerobic" *RQ* (corrected for changes in body CO_2 stores due to changes in LA production and mixed venous CO_2 content) would support the view that at these high levels of exercise the body changes from a carbohydrate/fat metabolic mixture to the almost exclusive use of carbohydrate as an energy source. The results are in accord with the view that the availability of carbohydrate (glycogen) is the major limiting factor to exercise of high intensity and long duration.

Key words: Exercise — Free Fatty Acid — Carbohydrate — "Aerobic" *RQ* — Lactic Acid — $\dot{V}_{O_2}$max.

It is now well established from in vitro animal experiments that muscles can utilise free fatty acid (FFA) as a fuel during exercise [15] but there have been fewer studies of this problem in intact organisms. The first workers to show that FFA rises during exercise in man were Basu *et al.* [2]. Their subjects performed light work ($\dot{V}_{O_2} \approx 1100$ ml/min) on a motor driven treadmill and after an initial fall, plasma FFA rose throughout the hour of exercise. However since this early study some workers have confirmed their results [8, 22] whilst some have found a fall [4, 13] and others no change [14] in FFA during work. Undoubtedly these discrepancies are in part due to different levels, duration and type of exercise used in the various studies and may be related to the findings of Issekutz *et al.* [19] on dogs. They clearly showed that a relationship exists between the production of lactic acid (and therefore the aerobic power of the dog) and the release and turnover of FFA during work. If their results can be applied to man they suggest that a relationship

may exist between change of FFA and relative work level (as per cent of maximum power output — $\dot{V}_{O_2}$max) which should hold throughout the operative range.

In the present study we have examined the relationship between the change in plasma FFA, Lactic acid (LA) production and aerobic power output of 7 healthy male subjects aged 21—41 years, over a wide range of exercise intensities up to and including maximum levels of work, on a motor driven treadmill. We have also attempted to relate our findings to the changes in metabolic mixture during work which can be inferred from changes in *RQ*. Such measurements of *RQ* and Metabolic mixture have often been criticised on the grounds that the CO_2 production ($\dot{V}_{CO_2}$) is not an accurate reflection of the metabolism of the muscle since a portion will arise from the change in body CO_2 stores. This is a particularly important factor at high levels of work where CO_2 will be displaced from tissue bicarbonate by *lactic acid* and a large change in CO_2 gas stores will take place due to the rise of mixed venous P_{CO_2} ($P\bar{v}co_2$). In order to overcome these difficulties we constructed a CO_2 balance [5, 6] for each exercise level and related our findings for the "aerobic" respiratory quotient of muscle to the changes in FFA.

Material and Methods

Seven healthy men were used as subjects for this study (Table 1). They were all carefully habituated to work [9] and reported to the Laboratory in the post-absorptive state. Each subject was studied on several different occasions during walking on the level and uphill on a treadmill for 1 hr; the aim being to span the whole range of their physical working capacity. The subjects rested for 1 hr before the commencement of the experiment and then were measured at rest and at 10 min intervals during exercise and the 2nd, 10th, 30th and 60th min of recovery. During the experiments the subjects inspired air through a Parkinson-Cowan (CD4) dry gas meter and expired into an 8 L. mixing chamber from which samples of mixed expired air were continuously sampled for determination of CO_2 and O_2 content using infra-red CO_2 and paramagnetic O_2 analysers. Blood was withdrawn

Table 1. *Physical characteristics of the 7 healthy male subjects*

Subject No.	Age (yr.)	Wt (kg)	Ht (cm)	$\dot{V}_{O_2}$max	
				l min^{-1}	ml kg^{-1} min^{-1}
1	38	68.1	174	3.00	43.9
2	21	69.5	178	2.86	41.2
3	37	67.4	180	2.80	41.4
4	27	88.7	188	3.60	40.5
5	26	71.1	179	3.00	42.2
6	31	61.1	178	2.90	46.5
7	41	72.4	176	3.02	41.8

from the ante-cubital vein on 3 occasions during rest and at the times stated above in exercise and recovery. The samples were analysed for LA, using a modification of the enzymatic method described by Gercken [11] and for FFA using the method of Duncombe [10]. Mixed venous PC_{O_2} was measured at rest and during exercise, immediately following the determination of oxygen intake ($\dot{V}_{O_2}$) and sampling of blood, using a rebreathing method [20] and the criteria previously described [12] if a $P\dot{V}_{CO_2}$ plateau was not obtained on the first occasion. In a small number of experiments the subjects also performed downhill walking [7]. These results are presented in Fig. 1.

Results

Time-Course of Plasma FFA and LA Changes in Exercise

Light and Moderate Exercise

During the transition from rest to light ($\dot{V}_{O_2} \approx 1.0$ l/min) and moderate ($\dot{V}_{O_2} \approx 1.5$ l/min) exercise there was a transient fall of plasma

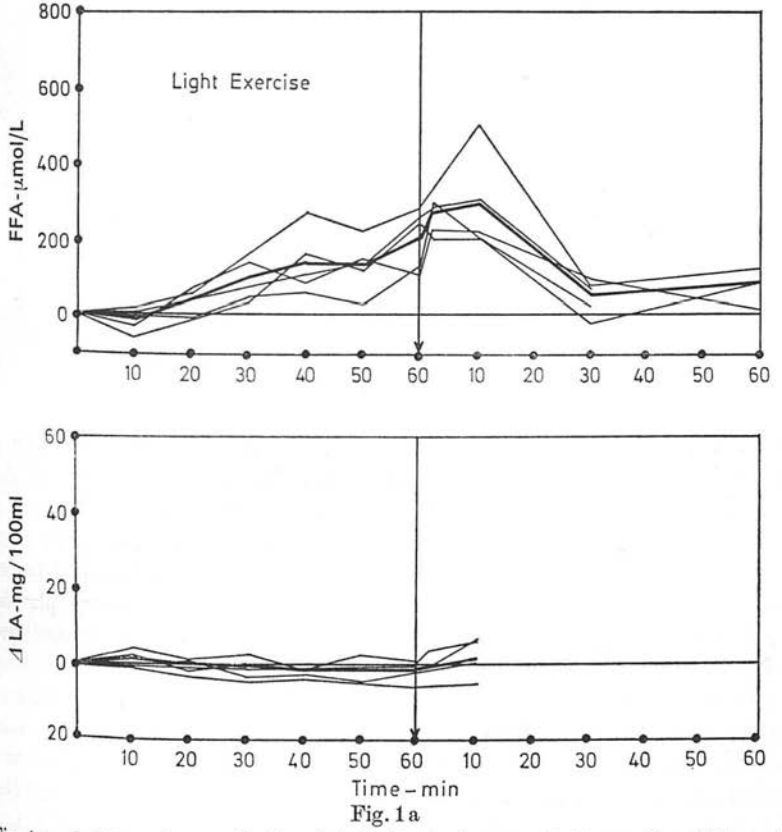

Fig. 1a

Fig. 1a—d. Mean (heavy line) and individual changes of plasma Free fatty acid (*FFA*) and lactic acid *(LA)* during: (a) light, (b) moderate, (c) light negative (eccentric) and (d) heavy exercise and recovery for the 7 subjects studied. The arrow indicates the end of exercise

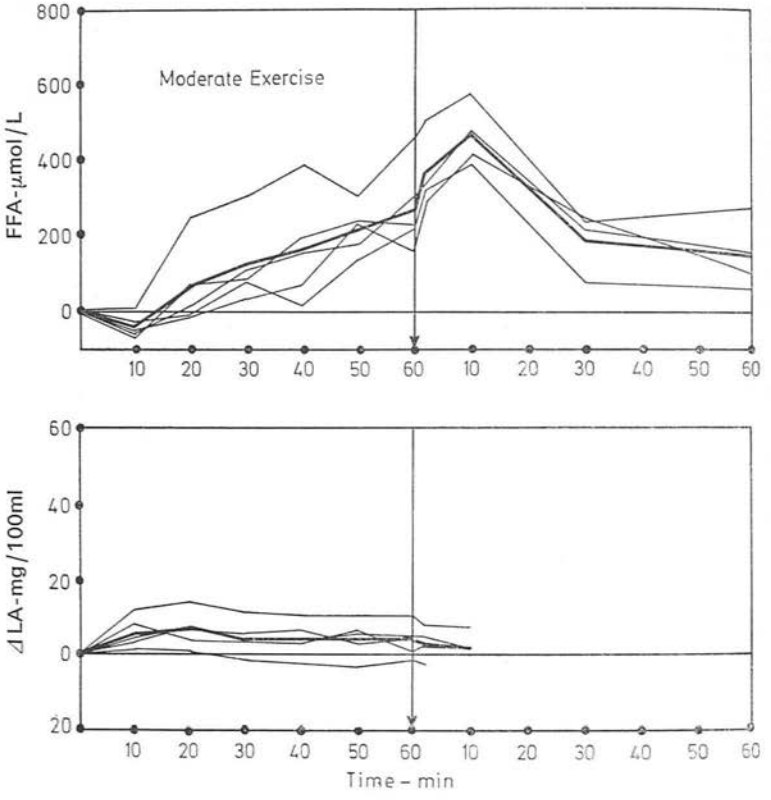

Fig. 1b

FFA. But from the 10th min plasma FFA invariably rose to reach a maximum value at the 60th min of work (Fig. 1). Immediately on the cessation of exercise plasma FFA again rose and showed a marked overshoot; the maximum recovery value being reached at the 10th min, thereafter plasma FFA decreased slowly to a new but enhanced (compared with the pre-exercise) baseline. After 1 hr of recovery plasma FFA remained elevated approximately 175 μmol/l above pre-exercise values.

Blood LA showed similar but opposite changes to plasma FFA. Immediately at the onset of exercise LA showed a small rise at the 10th min but thereafter declined and then remained constant throughout the work period. At the end of exercise LA decreased to resting levels at the 10th min.

During negative work ($\dot{V}_{O_2} \approx 1.0$ l/min) the changes in plasma FFA and LA were closely similar to those given above except that at the onset of exercise the transient depression in FFA was not observed. During

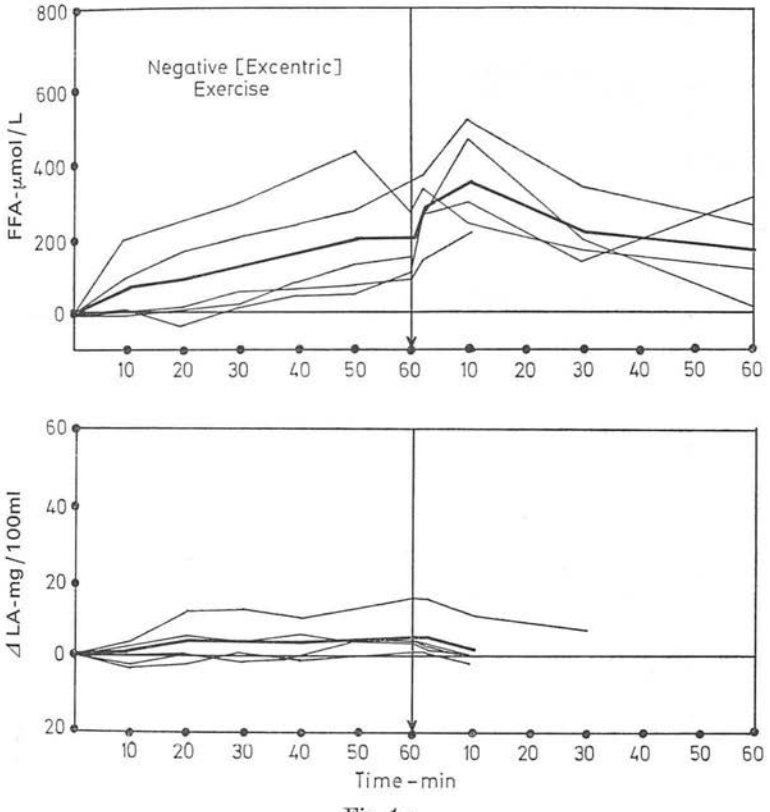

Fig. 1c

this period of initial rise in FFA, LA remained unchanged from resting values.

Heavy Exercise

During heavy exercise ($\dot{V}_{O_2} \approx 2.0$ l/min) plasma FFA concentration remained depressed at the onset of work until the 30th min thereafter a small rise in FFA was observed (Fig. 1). At the cessation of work there was again a sharp increase in FFA; a maximum concentration being reached at the 10th min of recovery. At the 60th min of recovery FFA remained elevated 220 µmol/l above the pre-exercise level.

Blood LA concentration showed qualitatively similar changes to those described for moderate work. Following an initial rise at the commencement of work LA concentration remained constant from the 20th—60th min and during the post exercise period immediately declined.

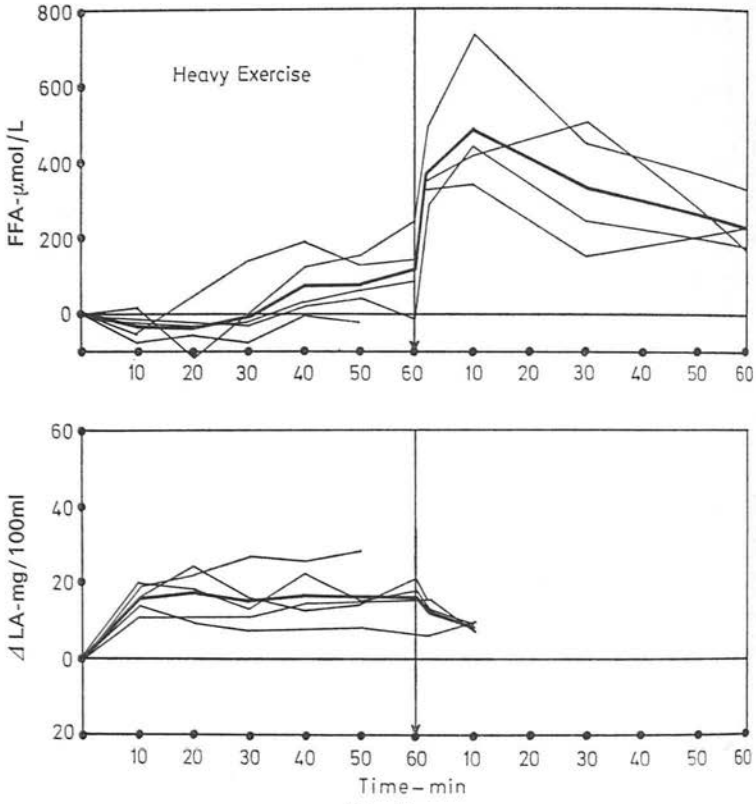

Fig. 1 d

Relationship of FFA to LA Concentration and $\dot{V}_{O_2}$ max

The relation between FFA and LA is shown in Fig. 2. For a given individual there appears to be an inverse linear relationship between the two variables; a rise in blood LA is associated with a corresponding fall in FFA concentration. In Fig. 3 the per cent rise and fall of LA and FFA is plotted in relation to relative work level (expressed as a per cent of $\dot{V}_{O_2}$ max) for the 7 subjects studied. It will be seen that up to a work level corresponding to $\sim 65\%$ $\dot{V}_{O_2}$ max; LA and FFA remain fairly constant, thereafter there is a marked fall in FFA, corresponding to a rise in LA.

The Aerobic RQ and Metabolic Mixture during Exercise

It is clear from Table 2 that the actual changes in CO_2 gas stores due to LA production and $\triangle P\bar{v}_{CO_2}$ are small and insignificant at the 60th min of exercise. The major changes in these two parameters occur

Table 2. *The change in blood lactic acid (LA) concentration and mixed venous P_{CO_2} during the 50—60th min of exercise, observed ($\dot{V}_{CO_2}$ obs) and corrected (for changes in body gas stores — $\dot{V}_{CO_2}$ corr) carbon dioxide output, observed oxygen intake ($\dot{V}_{O_2}$ obs) and observed and corrected RQ, in light and heavy exercise*

	△ LA mg %	△ $P\bar{v}_{CO_2}$ mm Hg	$\dot{V}_{CO_2}$ obs. (ml kg^{-1} min^{-1})	$\dot{V}_{CO_2}$ corr. (ml kg^{-1} min^{-1})	$\dot{V}_{O_2}$ obs. (ml kg^{-1} min^{-1})	*RQ* obs.	*RQ* corr.
Light exercise							
	+3.52	—4.1	9.22	8.70	11.35	0,81	0.77
	—0.53	—0.5	11.00	11.00	13.20	0.83	0.83
	+1.15	—1.7	9.33	9.13	11.23	0.83	0.77
	—0.92	—0.2	7.51	7.56	9.75	0.77	0.77
	+0.10	—2.0	8.22	8.07	10.52	0.78	0.77
	—3.27	—2.1	12.77	12.86	16.67	0.74	0.77
	—2.40	—0.5	13.05	13.22	16.41	0.79	0.81
Mean	—0.74	—1.59	10.16	10.08	12.73	0.79	0.78
SD	±2.25	±1.36	±2.52	±2.30	±2.80	±0.01	±0.02
Heavy exercise							
	+2.43	—1.0	19.83	19.58	21.88	0.91	0.89
	+2.72	+2.0	21.19	21.13	22.78	0.93	0.93
	—3.91	0.0	24.61	24.88	25.81	0.95	0.96
	—0.97	+0.5	18.50	18.60	20.16	0.92	0.92
	+4.56	—5.5	26.03	25.33	23.75	1.10	1.07
Mean	+0.97	—0.8	22.03	21.90	22.87	0.96	0.95
SD	±3.38	±2.84	±3.19	±3.06	±2.11	±0.025	±0.07

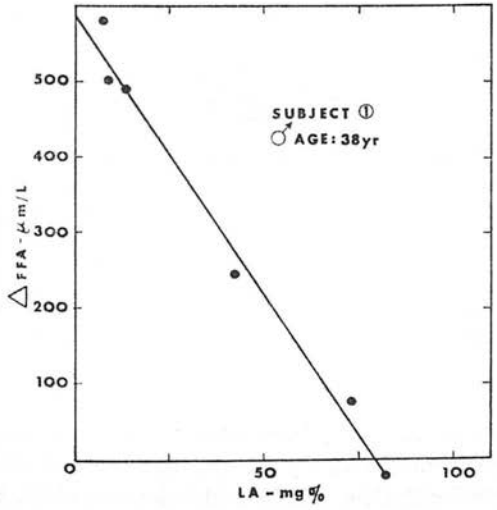

Fig. 2. Relationship between plasma free fatty acid (*FFA*) and lactic acid (*LA*)

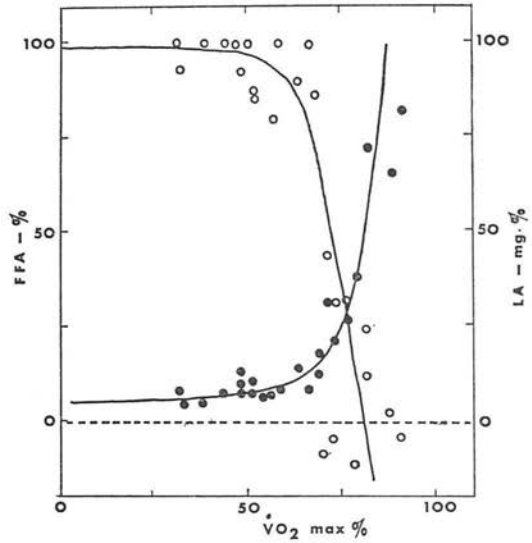

Fig. 3. Relationship of per cent change of plasma free fatty acid (*FFA*) and lactic acid (*LA*) to maximum aerobic power ($\dot{V}_{O_2}$max). o——o *FFA*; •——• *LA*

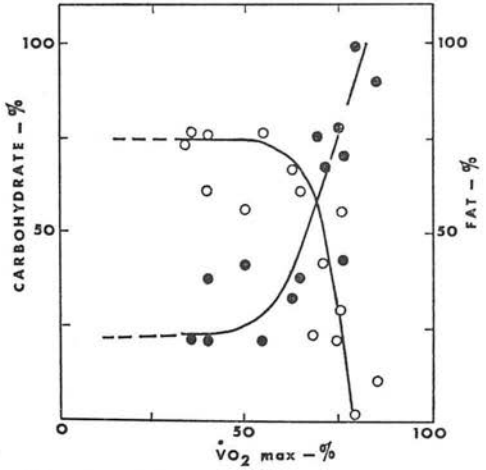

Fig. 4. Per cent fat and carbohydrate metabolism calculated from the "Aerobic" *RQ*, in relation to Maximum Aerobic power ($\dot{V}_{O_2}$max.). o——o % Fat; •——• % Carbohydrate

during the 1st 10 min of work, thus during exercise of long duration the "aerobic" *RQ* and observed *RQ* are synonymous and the latter may be taken as a "true" reflection of the fuel being metabolised. In Fig. 4 the per cent changes in fat and carbohydrate being metabolised are given

in relation to relative (as a per cent $\dot{V}_{O_2}$ max) work load. The pattern follows very closely to that given for FFA and LA (Fig. 3). At low levels of exercise a mixed (70% fat; 30% carbohydrate) fuel is utilized but beyond $\sim$ 65% $\dot{V}_{O_2}$ max the per cent fat falls rapidly and the body changes to the almost exclusive use of carbohydrate as a source of energy.

Discussion

The results of the present study confirm and extend to man the findings of Issekutz *et al.* on dogs. Namely, that there is a reciprocal relationship between plasma FFA and LA concentration in exercise and the changes in both these parameters are dependent on the maximum Aerobic power ($\dot{V}_{O_2}$ max) of the individual. At levels of exercise beyond $\approx$ 65% $\dot{V}_{O_2}$ max, plasma FFA decreases rapidly and blood LA rises. If one accepts the concept of Armstrong *et al.* [1] and others [17, 21] that the rate of release of FFA from adipose tissue controls the plasma FFA which in turn governs the rate of FFA uptake by the mass action effect, one can only conclude that there is marked reduction in FFA utilisation (due to suppression of release by LA [18] and a move towards the utilization of carbohydrate (CBH) for energy requirements, as exercise exceeds a critical level. The measurements of "Aerobic" *RQ* and related calculations of the changes in metabolic mixture in this study (Table 2 and Fig. 4) would fully support this thesis. Beyond $\approx$ 65% $\dot{V}_{O_2}$ max an individual changes rapidly from a mixed (70% fat: 30% carbohydrate) to an almost exclusively carbohydrate metabolic mixture (Fig. 4). During light exercise following a transient fall the plasma FFA concentration rises rapidly and during recovery falls with little or no marked overshoot (Fig. 1). It is worthy of note that the small transient decrease in FFA is not seen in light negative (eccentric) work which may be a reflection of the different blood flow changes which occur at the beginning of exercise in this type of work [7] and the absence of a significant energy contribution from anaerobic mechanisms (Fig. 1, and see also Davies and Barnes [7]). In contrast during heavy exercise plasma FFA falls and remains below resting levels for 50% of the exercise period. In the immediate post exercise period, FFA rises rapidly to levels compatible with those found during work at the lighter exercise levels (Fig. 1). Again if we accept the thesis of Armstrong *et al.* [1] then these results can only be interpreted as indicating during light exercise, an increased utilization of FFA slightly below its rate of release, giving rise to slow increase in plasma FFA. During heavy exercise the rate of release and utilization of FFA is suppressed possibly due to the increased involvement of anaerobic processes with subsequent incomplete breakdown of carbohydrate (glycogen) stores and a concomitant rise in LA within the muscle to

meet the energy requirements of the work. It will be noted however that although the rate of FFA utilization falls in heavy exercise it probably remains above the rate of release until the 30th min hence one observes a fall followed by slow rise in plasma FFA from the 30th—60th min of work. At the cessation of exercise when utilization probably diminishes and release of FFA may be enhanced a large rise in plasma FFA is again observed.

The individual variations in plasma FFA which occur in Fig. 1 are clearly accounted for in terms of work load. When the change in plasma FFA is related to relative (as per cent $\dot{V}_{O_2}$ max) rather than absolute $\dot{V}_{O_2}$ the picture is clear and consistent; beyond 65% $\dot{V}_{O_2}$ max, a fall in plasma FFA, a rise in anaerobic metabolism and increased utilization of carbohydrate (Figs. 3 and 4). These results are entirely in accord with the recent Swedish [3, 16] findings which have shown conclusively that at high rates of work the ability of an individual to continue exercise is purely dependent on the availability of stored glycogen: once this is exhausted exercise must cease. Clearly in absolute terms the athlete will be able to exercise at seemingly high exercise levels (compared to a sedentary subject) and draw upon a large reserve of FFA thus conserving his limited muscle glycogen — but in relative terms the physiological responses of the trained and untrained in terms of energy release and utilization will be identical. This is a strong argument for measuring the $\dot{V}_{O_2}$ max of an individual in all human studies aimed at elucidating metabolic control mechanisms. Similar arguments could be applied to thermal [8, 24] and cardiovascular [23] physiology.

Finally this investigation would also carry some implications for those concerned with the use of exercise in the treatment of obesity. For some patients, especially women, even walking at 3.5 mph on the level will be in excess of 60% of their $\dot{V}_{O_2}$ max. Thus the contribution of fat (from adipose tissue) as an energy source will be small. If exercise is combined with dietary measures then the patient should be encouraged to be active at or below 50% of his $\dot{V}_{O_2}$ max. This may involve extremely light work (in absolute terms) of extended duration.

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Ethnic differences in physical working capacity

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DAVIES, C. T. M., CAROLYN BARNES, R. H. FOX, R. OLA OJIKUTU, AND A. S. SAMUELOFF. *Ethnic differences in physical working capacity.* J. Appl. Physiol. 33(6): 726–732. 1972.—One hundred seventy-seven male and female subjects, aged 18–30 years, from four distinct ethnic groups—Europeans, Yoruba (Nigerian), and Kurdish and Yemenite Jews—have been studied during submaximal and maximal work on a stationary bicycle ergometer. The results show that the responses to exercise were essentially similar in the four groups, although there was a marked sex difference and the Kurds and Yemenites appeared to have a higher $\dot{V}_{O_2}$ for a given work output than the Europeans and Yoruba. The males in particular were able to reach similar values of maximum ventilation, cardiac frequency, and aerobic power output. When standardized for body size and composition (ml $kg_{lean\ body\ mass}^{-1}$ min^{-1}) the $\dot{V}_{O_2\,max}$ for each paired group (Caucasian-Yoruba; and Kurd-Yemenite) was identical. It was concluded that if differences in aerobic power output exist between ethnic groups, they must be small and therefore of doubtful biological significance—they are certainly less than the large intersubject variations which exist between members of the same race.

maximum aerobic power; submaximal exercise responses; lean body mass

GROUP DIFFERENCES in physical working capacity (PWC) in an otherwise genetically homogenous population have been repeatedly demonstrated (3, 4) but whether genuine population (ethnic) differences in PWC, which might have occurred through evolutionary natural selection, exists, is a matter of contention.

The present investigation is concerned with studies on four ethnically distinct populations: Caucasian, West African natives of Yoruba stock, and Kurdish and Yemenite Jews. The latter two groups are of particular interest; though genetically dissimilar they now live under the same environmental and ecological conditions as each other and thus their results are not confounded by present day (short-term) environmental changes in PWC. They form part of a large population study of Yemenite and Kurdish Jews living in the Negev region of Israel (20).

MATERIAL AND METHODS

The physical characteristics and ages of the 177 male and female subjects studied are given in Table 1. The 47 Caucasian (16 female and 31 male) subjects were of British stock and sedentary habit. The majority (8 female and 39 male) of the Yoruba investigated were of similar habit and therefore these groups can be directly compared. In addition a group (13 male) of active Yoruba workers from a Lagos glass and rubber factory and a small group of (8 male) villagers from the interior, who still practiced a traditional way of life, have been included. The Kurdish and Yemenite Jews were mainly farmers living in the Negev region of Israel.

All subjects were measured in a similar way with the same apparatus. They reported to the laboratory after a light breakfast or lunch. Each subject was required to rest for 15 min while the necessary electrodes were fitted and the details of the experiments were explained. The subject was then allowed to exercise on the bicycle ergometer for a period of 5–10 min while he became accustomed to breathing through the mouthpiece and habituated to the work procedure, after which he rested for a further 15 minutes.

The male subjects were then studied at four submaximal work loads: 300, 600, and either 750 and 900 or 900 and 1,200 kpm min^{-1}. A "continuous" form of exercise test was given, the work load being raised at the end of each 5-min period. The female subjects exercised in a similar way at work loads adjusted to suit their proportionately lower aerobic power outputs. During the exercise all subjects breathed through a low-resistance Otis-McKerrow valve via a short length of smooth 1-inch internal bore tubing into a series of vinyl plastic Douglas bags. In the Israel experiments, the Douglas bags were replaced by a Tissot spirometer and the oxygen and carbon dioxide meters by the Haldane apparatus. During the final 2 min at each work load, the cardiac frequency was taken from a standard ECG recorder, respiratory frequency was counted, and expired gas was collected. The expired gas was subsequently emptied through a Parkinson-Cowan CD4 gas meter and the oxygen and carbon dioxide concentrations measured using a paramagnetic oxygen meter (Beckman E_2) and either an infrared carbon dioxide meter (Beckman LB1) or a Katherometer (Cambridge Instruments), in a manner previously described (9). All the meters were calibrated at frequent intervals using gases of known (by the Haldane chemical method) concentration.

Oxygen intake ($\dot{V}_{O_2}$) was computed and regression equations for each subject and for the group as a whole were obtained for cardiac (f_H) and respiratory (f_R) frequencies

and ventilation ($\dot{V}_E$) volume on $\dot{V}_{O_2}$ and work load ($\dot{W}$). This enabled responses of the submaximal test to be expressed in terms of either a given $\dot{V}_{O_2}$ of 1.5 l min^{-1} (see Cotes et al., 8) or a standard $\dot{W}$ (900 kpm min^{-1}).

Following the submaximal test, the subjects were allowed to rest for 30 min and their maximum oxygen intake ($\dot{V}_{O_2\,max}$) and responses to maximum work were determined after the method described by Davies (9) and Shephard et al. (21). The criterion for maximal values was that $\dot{V}_{O_2}$ showed no further rise with increasing $\dot{W}$, plateau values to agree within ±5%.

In addition to the exercise tests various ancillary measurements were taken. These included forced vital capacity (FVC) by standard (McDermott) spirometry after the method described by Cotes (7), skinfold thickness at three sites (triceps, subscapular, and suprailiac), and limb volume measured by physical anthropometry (15).

RESULTS

Physical Characteristics

The Yoruba, Yemenite, and Kurdish males are significantly lighter in weight than their Caucasian counterparts (Table 1). This difference can partly be explained in terms of body fat. When expressed as kilograms of lean body mass (LBM) the weights of the six groups move closer together and a different pattern emerges. The Caucasians have similar LBMs to all groups measured ($P > 0.1$). However, the LBMs of the Kurds and Yemenites are significantly less than the Yoruba and the villagers.

The female subjects (Table 1) show a different pattern to that described for the males. The Kurdish and Yemenite women are significantly shorter than Caucasian and Yoruba, but their weights are the same. However, when allowances are made for body fat the Kurds and Yemenites emerge with significantly less LBM than either of the other two groups.

Responses to Submaximal Work Loads

The responses to submaximal exercise are summarized in Table 2.

Oxygen intake at a given work load. No significant differences in oxygen intake ($\dot{V}_{O_2}$) at a given work load ($\dot{W}$) of 900 kpm min^{-1}($\dot{V}_{O_{2(900)}}$) exist between the female groups. However, in the males, $\dot{V}_{O_{2(900)}}$ is significantly higher ($P < 0.01$) in all other groups when compared with the Caucasians with the exception of the small group of Yoruba villagers (Table 2).

Ventilation at a given oxygen intake. The ventilation at a given $\dot{V}_{O_2}$ of 1.5 l min^{-1}($\dot{V}_{E_{1.5}}$) in the males is significantly higher for the Yoruba compared with all other groups and similarly when the Yemenites and Kurds are compared with the Caucasians. The higher minute ventilation is almost entirely due to increased rate of breathing (f_R), V_T remaining constant for a given $\dot{V}_E$ in all groups. A similar pattern is shown among the female groups where the Caucasians have a lower $\dot{V}_{E_{1.5}}$ than all other groups although this only reaches conventional levels of significance in comparison with the Kurds ($P < 0.05$). The reason for this discrepancy is not clear, although it may be a direct result of residual anxiety and apprehension.

TABLE 1. *Age, weight, height, and lean body mass of male and female subjects*

	Group		Age (yr)	Weight (kg)	Height (cm)	L B M (kg)
MALES	Caucasian	(1)	23.77**2	72.26*4,5 **2,3 *6	175.00*6 **2,3,4 5	49.75
	n = 31		±2.90	±11.31	±7.01	±7.89
	Kurds	(2)	26.42	64.29	168.96**3	47.46*5
	n = 19		±3.63	±6.23	±6.75	±6.42
	Yemenite	(3)	25.25	63.79	163.20**4 **5,6	45.27**5 *4
	n = 20		±4.22	+10.20	±5.05	±8.67
	Yoruba(inactive)	(4)	25.76	61.59	169.95	50.25
	n = 33		+3.21	+9.16	±5.95	±6.27
	Yoruba (active)	(5)	25.13	61.64	169.57	51.46
	n = 23		±1.76	+5.89	±5.36	±4.68
	Yoruba(villagers)	(6)	25.28	61.63	168.50	52.05
	n = 7		±3.15	±4.55	±4.58	±4.92
FEMALES	Caucasian	(1)	23.80**4	54.52	160.19***2,3	39.95***2,3
	n = 16		±1.90	±6.32	±5.09	±1.87
	Kurds	(2)	25.50**4	58.65	153.89**4	32.33**4
	n = 12		±3.09	±10.85	±3.89	±6.09
	Yemenites	(3)	24.33	51.07	152.63*4	28.59*4
	n 12		±3.97	±10.50	±6.79	±5.31
	Yoruba	(4)	21.50	53.31	160.52	41.50
	n = 8		±1.85	±7.83	±4.92	±5.07

Values are means ± SD. Significance: * $P < 0.05$. ** $P < 0.01$. *** $P < 0.001$.

Cardiac frequency. In the males the cardiac frequency at a given $\dot{V}_{O_2}$ of 1.5 l min^{-1} ($f_{H(1.5)}$) is similar when Kurds are compared with Yemenites and Caucasians with Yoruba (Table 2). However, as might be expected, there were differences ($P < 0.05$) between both the Kurds and Yemenites when compared with the Caucasians and inactive Yoruba, and between the latter group and the active Yoruba. In the females the $f_{H(1.5)}$ is significantly higher ($P < 0.001$) than the males, but again the groups are similar. The Kurdish, Yemenite, and Yoruba female groups have statistically identical values although the mean $f_{H(1.5)}$ of the Caucasian women is significantly lower ($P < 0.01$) than the Kurds (173 ± 13.6 and 188 ± 20.8 beats min^{-1}, respectively).

Responses to Maximal Work Loads

The responses of the four ethnic groups to maximal work are summarized in Table 3. In absolute terms for the male subjects we see that the values for the maximum aerobic power ($\dot{V}_{O_2\,max}$), except for the inactive Yoruba (2.9 ± 0.48 l min^{-1}), are essentially similar and range from 3.24–3.39 l min^{-1}.

A similar pattern is shown for maximum cardiac frequency. The highest group mean value for $f_{H\,max}$ recorded was 193 ± 6.7 beats min^{-1} in the Caucasians and the lowest 187 ± 7.2 in the Yemenite Jews, a difference of 6 beats min^{-1}.

In contrast to the above the maximum mean values of ventilation were less uniform. The highest values were seen

TABLE 2. *Responses to exercise*

	Group		$\dot{V}O_{2\ 900}$ L.min.$^{-1}$ S.T.P.D.	$\dot{V}_{E\ 1.5}$ L.min.$^{-1}$ B.T.P.S.	$f_{H\ 1.5}$ beats min^{-1}	Predicted maximum aerobic power ($\dot{V}O_2$max) L.min.$^{-1}$	ml.kg$_{(wt)}^{-1}$min^{-1}	ml kg$_{(LBM)}^{-1}$min^{-1}
MALES	Caucasian	(1)	2.06$^{**2,3,5}_{**4}$	37.68$^{***4,5,6}_{**2}$ ^{*3}	136 ^{*2,3}	2.95^{**5}	40.82$^{***5}_{**3,4,6}$	59.34$^{**3}_{*5}$
	n = 31		±0.11	±4.44	±14	±0.44	±6.80	±10.68
	Kurds	(2)	2.28^{*6}	42.01^{***4,5,6}	128 ^{*4}	2.89^{**5}	44.44^{***5}	60.13
	n = 19		±0.23	±6.62	±12	±0.46	±7.05	±10.20
	Yemenites	(3)	2.36$^{**4,6}_{*5}$	40.86^{***4,5,6}	127 ^{*4}	2.98^{*5}	46.91^{***5}	66.48^{***4}
	n = 20		±0.22	±4.18	±15	±0.50	±5.27	±7.07
	Yoruba (inactive)	(4)	2.18	51.87^{**6}	137 ^{*5}	2.81^{***5}	45.91^{***5}	56.18^{***5}
	n = 33		±0.19	±8.03	±13	±0.50	±7.26	±9.27
	Yoruba (active)	(5)	2.22^{*6}	51.41^{**6}	129	3.40	55.53^{*6}	66.25
	n = 23		±0.20	±7.09	±13	±0.58	±6.49	±10.02
	Yoruba (villagers)	(6)	2.06	60.93	131	2.99	48.50	61.23
	n = 7		±0.11	±9.28	±14	±0.38	±4.05	±4.04
FEMALES	Caucasian	(1)	2.07	45.82^{*2}	173 ^{**2}	1.89	34.55	48.17^{*3,4}
	n = 16		±0.47	±7.67	±14	±0.34	±6.79	±7.58
	Kurds	(2)	2.22	54.54	188	1.63	28.96	54.37^{*4}
	n = 12		±0.29	±5.23	±21	±0.33	±8.28	±10.70
	Yemenites	(3)	2.29	49.95	175	1.78	35.40	62.40^{**4}
	n = 12		±0.73	±6.76	±22	±0.43	±7.73	±15.63
	Yoruba	(4)	2.15	52.55	183	1.69	31.60	40.52
	n = 8		±0.44	±8.90	±21	±0.30	±3.65	±5.12

Oxygen intake at a given work load of 900 kpm min^{-1} ($\dot{V}O_{2(900)}$); ventilation ($\dot{V}E_{1.5}$) and cardiac frequency ($f_{H(1.5)}$) at an $\dot{V}O_2$ of 1.5 l min^{-1} and predicted maximum aerobic power ($\dot{V}O_{2\ max}$) in absolute (l min^{-1}) and relative (ml kg^{-1} (weight) min^{-1} and ml kg^{-1} (LBM) min^{-1}) terms. Values are means ± SD. Significance as in Table 1.

TABLE 3. *Directly measured maximum oxygen intake ($\dot{V}O_{2\ max}$), ventilation ($\dot{V}E_{max}$), and cardiac frequency ($f_{H\ max}$)*

Group		$\dot{V}O_{2\ max}$ L.min.$^{-1}$ S.T.P.D.	$\dot{V}_{E\ max}$ L.min.$^{-1}$ B.T.P.S.	$f_{H\ max}$ beats min.$^{-1}$
Caucasian	(1)	3.39^{*4}	133.14^{***2,3}	193 ^{**3}
n = 31		±0.43	±18.04	±7
Kurds	(2)	3.24	111.49^{**5}	193
n = 12		±0.38	±13.75	±6
Yemenites	(3)	3.29	110.89^{**5}	187
n = 15		±0.70	±13.30	±7
Yoruba(inactive)	(4)	2.92	111.87	192
n = 6		±0.48	±24.01	±4
Yoruba (active)	(5)	3.28	133.95	190
n = 6		±0.40	±16.73	±8

Values are means ± SD.

in the Caucasian (133.1 ± 18.04 l min^{-1}) and active Yoruba (133.9 ± 16.7 l min^{-1}). The remaining three groups have mean values some 20 l min^{-1} lower, although due to the large intersubject variation the only differences of $\dot{V}E_{max}$ which reach conventional levels of significance are those between the two Jewish groups and the Caucasian and active Yoruba subjects. These differences reflect in part the willingness of the subjects from the respective groups to force themselves to exercise at levels of work beyond which the $\dot{V}O_{2\ max}$ occurs and are in this sense independent of the absolute values of $\dot{V}O_{2\ max}$ recorded.

Unfortunately, the female subjects of all groups were more reluctant than the men to exercise at maximal effort; only 20 (14 Caucasians, 2 Kurds, and 4 Yemenites) were willing to cooperate in a maximum test. It is therefore difficult to draw conclusions from the results obtained. However, the data do suggest that the absolute values for all parameters except $f_{H\ max}$ are significantly lower than those found for the men. The mean $\dot{V}O_{2\ max}$ ranges from 1.5 to 2.2 l min^{-1}, approximately two-thirds of the value found for men, and the $\dot{V}E_{max}$ is in the range 60–78 l min^{-1}.

Prediction of Maximum Aerobic Power

The method of predicting $\dot{V}O_{2\ max}$ from measurements of f_H and $\dot{V}O_2$ taken at submaximal work loads has been primarily developed on Caucasian data from the original work of Åstrand and Rhyming (5). Thus, before applying this technique to other ethnic groups, one must be sure that they behave in a similar way to Caucasians (cf. Maritz et al. (17) for South African Bantu subjects). In particular, it must be shown that the $\dot{V}O_2/f_H$ relationship is linear within a certain range and all members of the populations to be compared can reach similar $f_{H\ max}$.

The results of this study (Table 3) would seem to indicate that comparison of these ethnic groups in terms of their predicted $\dot{V}O_{2\ max}$ is valid since they all appear to behave in a similar way to the Caucasians studied in the United Kingdom, the predicted underestimating the observed $\dot{V}O_{2\ max}$ due to the asymptotic nature of the $\dot{V}O_2/f_H$ curve by some 10% (see Davies, 9, and Fig. 1).

Table 2 summarizes the predicted values of $\dot{V}O_{2\ max}$ de-

rived from extrapolation of the $\dot{V}O_2/f_H$ relationship obtained at four submaximal work loads to a maximum cardiac frequency of 195 beats min^{-1} (cf. 9).

In absolute terms the $\dot{V}O_{2\,max}$ of the active male Yoruba (3.40 ± 0.58 l min^{-1}) is 0.4 l min^{-1} higher than any other group and this difference is significant ($P < 0.05$) except in the case of the small groups of villagers. However, for a valid comparison to be made some account must be taken of the differences in body stature and composition. If $\dot{V}O_{2\,max}$ is expressed in terms of total body weight, the Caucasians have a significantly ($P < 0.01$) lower $\dot{V}O_{2\,max}$ than all other groups except the Kurds, although when account is taken of variations in body fat this only remains true when the Caucasians (59.34 ± 10.68 ml kg_{LBM}^{-1} min^{-1}) are compared with the Yemenite and active Yoruba (66.48 ± 7.07 and 66.25 ± 10.02 ml kg_{LBM}^{-1} min^{-1}, respectively). It should also be noted that no difference exists in $\dot{V}O_{2\,max}$, however expressed, between the Kurds and Yemenites.

In the female groups, the $\dot{V}O_{2\,max}$ are essentially the same both in absolute terms and relative to total body weight. Only when the $\dot{V}O_{2\,max}$ is expressed in milliliters per $kilogram_{LBM}$ and minute do differences arise and then these are similar to those described for the men. Thus the Caucasians (48.2 ± 7.6) and inactive Yoruba (40.5 ± 5.1) have significantly lower values than the Kurds (54.4 ± 10.7) and Yemenites (62.4 ± 15.6), but the Kurd-Yemenite comparison shows no significant difference and that of Caucasian with Yoruba only a slightly higher value for the former.

It should also be noted that the consideration of the LBM of the subject significantly reduces but does not eliminate completely the sex difference between members of the same racial group.

Multiple Regression Analysis

Again it is of interest to know whether the multiple regression equations for the prediction of maximum aerobic power developed by Cotes et al. (8), which essentially contain a factor for the lungs, heart, and muscle mass involved in exercise, can be applied to different groups. To investigate this problem we have performed multiple regression analysis of forced vital capacity (FVC), cardiac frequency at $\dot{V}O_2$ 1.5 l min^{-1} ($f_{H(1.5)}$), and limb volume estimated from anthropometry (LV) on $\dot{V}O_{2\,max}$ for the Yoruba data. The resulting equation is of the form:

$$\dot{V}O_{2\,max} = 7.65 - 0.049\,FVC - 0.025\,f_{H(1.5)} - 0.08\,LV$$

By use of this formula the standard error of $\dot{V}O_{2\,max}$ is $\pm$ 316 ml; this represents a coefficient of variation (CV) of $\sim$10%. This figure is almost identical to that found for Caucasian data (8).

Distribution of Predicted Maximum Aerobic Power

Christensen (6) has given now generally accepted values of $\dot{V}O_2$ which correspond to moderate, heavy, and very heavy work in everyday life. In our terms they may be taken as 30, 40, or 50 ml kg body $weight^{-1}$, respectively. Considered in this light then all the male members of the four ethnic groups would be fit for sustained moderate work. However, for heavy work the picture changes: in the Caucasian group only 18 out of 31 (18/31 = 58%) males are fit for this type of occupation, compared with 35/38 (92%) Kurds and Yemenites, 27/33 (82%) inactive Yoruba, 22/23 (96%) active Yoruba, and 7/7 (100%) of the villagers. For very heavy work the comparisons are even more striking: 4/31 (13%) Caucasians, 9/38 (29%) Kurds and Yemenites, 2/33 (6%) inactive Yoruba, 17/23 (74%) active Yoruba, and 3/7 (43%) villagers.

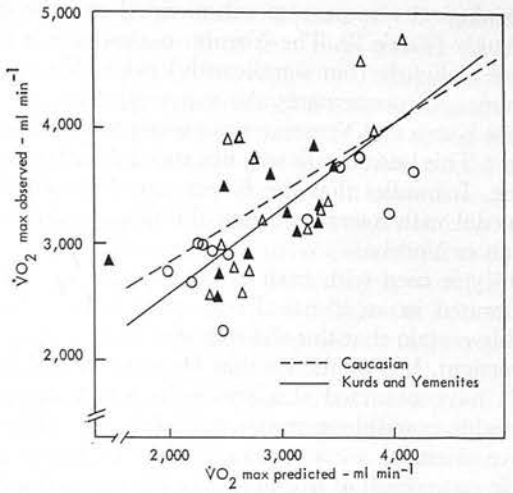

FIG. 1. Observed maximum aerobic power ($\dot{V}O_{2\,max}$) in relation to predicted $\dot{V}O_{2\,max}$ from $\dot{V}O_2$ and f_H measurements taken at four levels of work. Kurds ▲; Yemenites △; Yoruba ○.

DISCUSSION

The level of fitness of a population is dependent on many factors, e.g., an individual's age, nutrition, environmental conditions, and habitual activity, and when comparing racial groups to discover if true ethnic differences exist one has to be careful to see that the exercise test procedure given to each group is the same and variability due to techniques or observer error is kept to a minimum. In the present study only the Kurdish and Yemenite Jews meet these criteria completely in as much that they are two genetically distinct ethnic groups, now living under identical environmental conditions, and with levels of nutrition and habitual activity which are similar. They were tested by the same observer using one standard set of exercise equipment. The same is true but to a lesser extent of the inactive Yoruba and the Caucasian male and female groups. These groups are genetically dissimilar and live sedentary lives, but levels of nutrition and environmental conditions differ. The active Yoruba and villagers form two separate groups and cannot be used as a valid means of compairson. Their results, however, are of interest and serve as a reference point to other published studies in the literature. Thus, if comparison is made of Caucasians with inactive Yoruba and Kurds with Yemenites, one can examine the various responses to submaximal and maximal exercise and decide whether a true ethnic difference exists or not.

Submaximal Responses

Comparing only Kurds with Yemenites and Yoruba with Caucasian subjects there are few basic differences in

the physiological responses to submaximal exercise in the present study (Table 2). The Yoruba males have a higher $\dot{V}E_{1.5}$ and a slightly (but significantly) raised $\dot{V}O_{2(900)}$ than their Caucasian counterparts. An increased $\dot{V}O_{2(900)}$ is also seen in the Kurds and Yemenites as a group when compared to the rest. This latter result was unexpected and somewhat surprising. It implies that the Kurds and Yemenites, as a group, pedal with lower mechanical efficiency than either Caucasian or Yoruba.

The bicycle used with each group was the same and it was calibrated in an identical way and we are therefore reasonably certain that this did not arise due to some technical oversight. It is of interest that Hermansen and Anderson (12) have obtained similar results when comparing athletic with nonathletic males and thus the differences may have arisen as a consequency of the variation in the anaerobic component of work (11). In the main, the Kurds and Yemenites were active agricultural workers, whereas the Caucasians were sedentary. At the higher work loads, the Caucasians would be pedaling at a higher relative $\dot{W}$ (considered as a percentage of $\dot{V}O_{2\,max}$) and, therefore, it is conceivable that in this group an increasing amount of exercise is performed anaerobically and thus the oxygen cost would be diminished. Indeed, as one would expect if this account if correct, the active Yoruba also have a higher $\dot{V}O_2$ at a given $\dot{W}$ than inactive members of the same or Caucasian groups, although this only reaches significant levels in the case of the latter group and the villagers ($P <$ 0.001 and 0.05, respectively). However, it should be noted that, although a theory based on the consequences of the variation in anaerobic component of work might explain the differences between the Kurds and Yemenites and the others, it does not account for the difference observed between the Caucasian and inactive Yoruba males.

Maximum Responses

The results of this study show conclusively that Yoruba, Kurds, and Yemenites can reach similar maximum cardiac frequencies, ventilation, and oxygen intake to Caucasians (Table 3). Indeed, $f_H/\dot{V}O_2$ curves behave during exercise in an exactly similar way to that described previously for the latter group (9). Thus the extrapolation procedures for the prediction of $\dot{V}O_2$ which have been developed primarily on Caucasian subjects may be applied to the different ethnic groups in this study.

In absolute terms the predicted $\dot{V}O_{2\,max}$ is similar for all the male groups except, as might be expected, for the active Yoruba group. However, Caucasian males are heavier than their Yoruba, Yemenite, and Kurdish counterparts, due mainly to an increased amount of body fat (Table 1 and cf. di Prampero and Cerretelli, 18). Thus in relative terms of milliliters per kilogram$_{wt}$ and minute, though no significant differences in $\dot{V}O_{2\,max}$ exist between Yemenites and Kurds, the Caucasians have a slightly lower ($\backsim$10%) value than the inactive and village Yoruba. This difference disappears, however, when $\dot{V}O_{2\,max}$ is expressed in milliliters per kilogram$_{LBM}$ and minute.

Among the female groups the Kurds and Yemenites show a significant difference to the Yoruba and Caucasians when $\dot{V}O_{2\,max}$ is considered in LBM terms. In addition, the latter group demonstrate a slightly higher value than the Yoruba (48.17, cf. 40.52 ml min^{-1} kg LBM^{-1}). However, when expressed in absolute terms or relative to total body weight, all groups show closely similar values.

Thus it would seem that provided the groups are comparable (as in the Kurdish-Yemenite and Caucasian-Yoruba comparisons of this study) there is little evidence to support the existence of racial differences in PWC of man. Further, it is our view that such evidence that has been adduced in the past for ethnic differences can be explained in terms of environmental (cf. inactive-active Yoruba groups of this study) rather than racial factors. The only other study we

TABLE 4. *Comparison of predicted maximum aerobic power ($\dot{V}_{O2\,max}$) of various ethnic groups*

	Ethnic Group	$\dot{V}O_{2\,max}$ ml kg^{-1} min^{-1}	Reference
MALES	**Primitive**		
	Arctic Indian	49.6	Andersen et al (2)
	Bushman	47.9	Wyndham (24)
	Dorobo and Turkana	46.0	di Prampero & Cerretelli (18)
	Pygmies	47.4	Andersen (personal communication)
	Yoruba (villagers)	48.5	Present data
	"Inactive"		
	Bantu	44.6+*	Wyndham et al (25)
	Caucasian		
	American	43.5*	Taylor et al (23)
	British	41.0	Present data
	Canadian	44.5*	Shepherd et al(20)
	Czechoslovakian	40.6*	Skranc (22)
	German	40.5*	Konig et al (16)
	Italian	45.0	di Prampero & Cerretelli (18)
	Norwegian	44.0	Hermansen and Andersen (12)
	South African	43.5+*	Wyndham et al (25)
	Ethiopian	39.9	Andersen (personal communication)
	Japanese	45.0*	Ikai and Shindo (14)
	Kurds	44.4	Present data
	Pascuans	45.4	Andersen (1)
	Yemenites	46.9	Present data
	Yoruba	45.9	Present data
	"Active"		
	Caucasian		
	Swedes	52.0	Åstrand (4)
	Lapps	54.0	Andersen (1)
	Yoruba	55.5	Present data
FEMALES	**Inactive**		
	Caucasian		
	British	39.6	Present data
	Czechoslovakian	29.5*	Skranc (22)
	Swedish	35.9*	Åstrand (3)
	Japanese	33.0*	Ikai & Shindo (14)
	Kurds	29.0*	Present data
	Pascuans	31.0	Andersen (1)
	Yemenites	35.4	Present data
	Yoruba	31.6	Present data
	Active		
	Caucasian		
	Swedish	43.6*	Åstrand (4)
	Lapps	42.0	Andersen (1)

Males and females aged 18–30 years.

are aware of in which the environmental (including habitual activity) conditions were exactly similar is that of Robinson et al. (19) on white and American negro sharecroppers. They found that the two groups had virtually the same maximum aerobic power output (49.6 ± 0.51 against 49.9 ± 0.52 ml kg^{-1} min^{-1}).

Those in favor of ethnic differences in PWC have often argued on the basis of primitive man's need to be active in order to survive. It is this factor, they state, which probably conferred a special advantage on the species and through the process of natural selection produced populations of differing physical and physiological characteristics dependent upon the degree of climatic and ecological stresses within a given environment. The critical question to ask, therefore, is whether the basic premise is correct: was man indeed more active in the past than he is today? One way we can attempt to answer this question at this late date is to compare the fitness and habitual activity patterns of some primitive populations still existing today with modern civilized man.

In the present study only one group falls into this former category—the Yoruba villagers. It will be seen that the level of their $\dot{V}O_{2\,max}$ is in no way exceptional and differs little from that given for sedentary Caucasian man (Table 4). This result is also supported by studies on primitive man which exist in the literature and are summarized in Table 4. Close inspection of this table reveals that the primitive populations are in no way superior to other more "advanced" groups. The Kalahari Bushmen (reputed to be one of the most primitive groups remaining in the world today), for example, score only slightly higher than average sedentary Caucasian males. Indeed, if one observes these primitive groups one finds that their pattern of life is certainly hard but not necessarily active. They do have periods of intense activity when hunting and food gathering, but once the appetite has been satiated they spend long hours either sleeping or sitting around the communal fire. It is likely that every society throughout its existence has exhibited these cyclic changes in physical activity and possessed some members who have exerted themselves more than others, hence the wide intersubject variability we see in physical working capacity, but from the evidence of this study interpopulation fitness would seem to remain at a fairly constant level.

One could argue, of course, that the test we are using as a measure of fitness is totally inadequate and the numbers in our sample too small to reach valid conclusions. In part, one must agree. Davies (10) has recently shown that in order to demonstrate a 5% difference in $\dot{V}O_{2\,max}$ between two populations means, for example, one needs to study at least 100 subjects in each group and clearly our population sizes fall short of this ideal. Similarly, the $\dot{V}O_{2\,max}$ as a test of PWC certainly falls short of the ideal; nevertheless it does measure one aspect of fitness, namely, the fitness to perform aerobic work at maximum levels. Thus until more sophisticated tests of PWC have been developed and larger numbers of subjects are studied, we cannot be sure whether differences in physiological performance exist, but from the evidence of this study such differences, if found, are likely to be small and certainly less than the wide variation in PWC between individuals of the same race.

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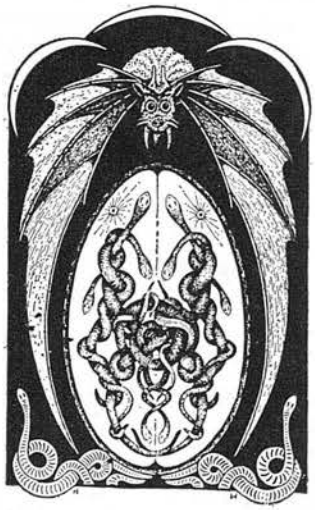

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Relationship of maximum aerobic power output to productivity and absenteeism of East African sugar cane workers

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Relationship of maximum aerobic power output to productivity and absenteeism of East African sugar cane workers

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Davies, C. T. M. (1973). *British Journal of Industrial Medicine*, **30**, 146-154. **Relationship of maximum aerobic power output to productivity and absenteeism of East African sugar cane workers.** The relationship of the physiological responses to laboratory exercise on an upright bicycle ergometer and predicted maximum aerobic power output ($\dot{V}O_{2max}$) to daily and season output has been investigated in 78 cane cutters aged 18 to 50 years working on the Kilombero sugar estate, Tanzania.

The results showed that the $\dot{V}O_{2max}$ was independent of absolute productivity in terms of kilotons of cane cut per season but positively correlated ($r = +0{\cdot}46$) with daily output. There was a small but significant ($r = -0{\cdot}32$, $P < 0{\cdot}001$) negative association of $\dot{V}O_{2max}$ with the number of days that an individual voluntarily absented himself from the cane fields. The importance of these findings to the selection and training of personnel for the cane-cutting industry are discussed. It was suggested that the industry might consider the introduction of a simple exercise (step) test together with a medical examination and simple anthropometric measurements at the time of recruitment of workers at the beginning of the season.

It has now been generally accepted that aerobic power output ($\dot{V}O_2$ max) measured in the laboratory provides a useful and valid guide to an individual's exercise tolerance (Shephard, Allen, Benade, Davies, Prampero, Hedman, Merriman, Myhre, and Simmons, 1968). Indeed, much time and effort has been spent in various laboratories throughout the world in the study of methods of measurement and the biological significance of $\dot{V}O_{2\ max}$. From these studies it is clear that the $\dot{V}O_{2\ max}$ essentially reflects the ability of the cardiorespiratory system and working muscles to transport and utilize oxygen and thus measures the capacity of the body for sustained work. It is also known that the $\dot{V}O_{2\ max}$ is affected by age, sex, physique, and training, and although none of these factors is in dispute, the vital question in the industrial situation as to whether the $\dot{V}O_{2\ max}$ is actually related to work output under realistic everyday conditions remains unanswered.

To my knowledge, the only attempt to study this problem has been that of Wyndham, Cooke, Munro, and Maritz (1964) in the gold mining industry in South Africa. They investigated 28 African miners who were daily involved in a heavy task loading rock underground. They found that although they could demonstrate significant differences in productivity and physical working capacity between the men in the group, no correlation existed between maximum aerobic power and physical performance. They concluded that psychological, rather than physiological, factors were the main determinants of productivity in the gold mine. Indeed, a later study (Wyndham and Cooke, 1964) showed that motivation, particularly in the

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form of the white European leadership, was the most important single factor in governing the output of the African worker.

Their study is of obvious importance to the field of industrial medicine but it is difficult to interpret on several counts. First, the numbers studied were small and the sample was not random but biased towards those subjects with high maximum aerobic power within a narrow range; 21 of the 25 subjects had $\dot{V}O_{2\,max}$ values in excess of the mean value found in their previous 'random' study of 350 Africans working in the gold mining industry in Johannesburg (Wyndham, Strydom, Morrison, Williams, Bredell, and Heyns, 1966). Secondly, the overall supervision of the African miners was being effected by Europeans and the output data were clearly affected by and related to this factor. The results of the investigation, therefore, cannot be directly applied to most developing and developed countries of the world where, particularly in the former case, the tendency has been to replace expatriate supervision of workers by indigenous personnel.

In the present study, the maximum aerobic power of 78 male African workers, aged 18 to 50 years, employed as cane cutters on a government-owned sugar estate in Tanzania, East Africa, has been investigated in relation to productivity (in terms of the daily amount of cane cut and earnings) and absenteeism throughout the six months of a cane-cutting season.

The study was conducted in two parts in a way similar to that originally described by Wyndham *et al.* (1964). In the first instance it was necessary to find a situation in which the productivity could be measured accurately, and to establish (1) whether large inter- and intra-subject variability existed in output, and (2) whether this variability was a random or consistent phenomenon enabling the high producers to be clearly identified. Once these two factors were established, the second part of the study was undertaken to investigate the relationship between physiological performance and cutting ability in the cane fields. At the same time an attempt was made to answer two subsidiary questions. Clearly, if it could be shown that productivity was related to certain physiological measurements in the laboratory then it would be important to devise a simple work capacity test which could be incorporated into any future selection training programme in the sugar industry. For this reason the cane cutters were given a simple exercise (step) test to perform at the time of their recruitment. In addition certain anthropometric measurements were taken at the beginning of May and again in December in order to assess the effects of cane cutting on the body size and composition of the workers during the season.

Procedure

The place chosen for the study was the Kilombero sugar estate, Kidato, Tanzania. The reasons for this choice were that the conditions at the estate offered an opportunity to measure not only the major physiological variables, but also the actual work performed in the cane fields for each individual worker in terms of tonnage of cut cane. The estate possessed an excellent medical service and small hospital, and careful records were kept of sickness and absenteeism from the cane fields. Both the unions and management of the estate cooperated wholeheartedly in the investigation and all subjects gave their informed consent before participating in the study.

The 585 sugar cane workers employed on the estate were first screened at the beginning of the season (Fig. 1) for age, weight, height, and blood haemoglobin (Table 1) and all were given a thorough medical examination, which included a simple work (stepping) capacity test. These measurements, except the step test, were routinely carried out each year at the estate at the beginning of the cane-cutting season. Subjects who were either outside the age range 18-50 years or had clinical signs of disease, which included severe parasitic infection and anaemia, were excluded. During this part of the study the inter-subject variability of the various parameters measured was established and records of the previous year's earnings were taken. Following the preliminary examination the cane cutters started to work on the estate and towards the end of the season (December) a further selection of subjects was made on the basis of output (tons of cane cut) and earnings from the beginning of the season. Fifty-eight young men aged 18 to 35 years were divided into three groups representing low, medium, and high outputs and earnings together with a group of 20 older men over 35 years of age; they were asked to attend the laboratory which for the purposes of the study had been

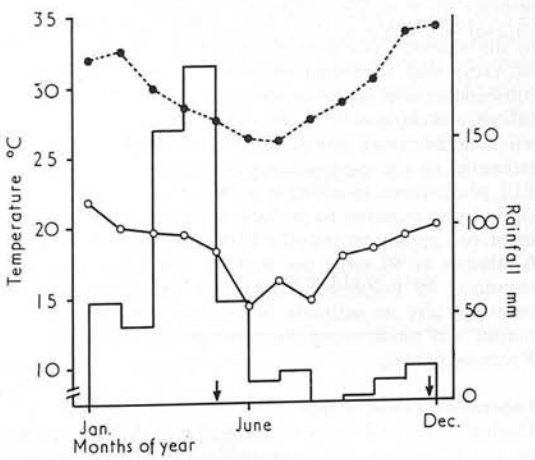

FIG. 1. Mean changes of maximum (● – – ●) and minimum (○ — ○) temperature and rainfall (open columns) at the Kilombero sugar estate for 1972. The arrows mark the dates of (1) the preliminary survey and (2) the definitive study.

TABLE 1

PHYSICAL CHARACTERISTICS OF A SAMPLE OF 285 CANE WORKERS FROM TOTAL LABOUR FORCE OF 585 MEN EMPLOYED IN CANE FIELDS

Age (yr)	*Weight (kg)*	*Height (cm)*	*Σsf (mm)*	*Circumference*		*Hb (g/100ml)*	$\dot{V}O_{2\ max}$ *(l.min⁻¹)*
				Arm (cm)	*Calf (cm)*		
26·8 ±4·7	62·2 ±5·3	165·9 ±5·2	23·2 ±4·0	26·2 ±1·5	33·7 ±1·9	11·8 ±1·2	2·92 ±1·23

Σsf = sum of 4 skinfold thicknesses

set up close to the cane-cutting fields, in order that they could be investigated in greater detail.

On the first occasion at the laboratory the details of the experiment were explained to the subjects and they became accustomed to pedalling the bicycle and breathing through the mouthpiece. A sample of blood was taken for the determination of haemoglobin and the subject's age, height, weight, skinfold thickness, and limb volume, by both physical anthropometry and water displacement, were taken.

On the second occasion the subjects were required to pedal the bicycle with at least four consecutive workloads of increasing severity up to or near maximal levels of performance.

Methods

Details of the laboratory methods used in this study have already been given (Davies, 1968 and 1972; Davies, Barnes, and Godfrey, 1972) and the reader is referred to these publications for further information; only a brief outline of the methodology involved will be given here.

The initial (screening) tests on the workers included measurements of *body weight* using a standard hospital (Avery) scale accurate to within ±250 g, and body height to the nearest millimetre using a stadiometer. *Skinfold thickness* was measured at four sites (biceps, triceps, subscapular, and suprailiac) using a Harpenden skinfold caliper, and *calf and biceps circumferences* were determined with a tapemeasure. *Haemoglobin* (Hb) concentration was estimated by the cyanmethaemoglobin method using an EEL photometer. In addition to these measurements each subject was required to perform a simple step test. This involved stepping on and off a 12 in (300 mm) high stool for 6 minutes at 30 steps per minute. The heart rate was measured by palpation during the first 10 seconds of recovery, and an estimate of maximum aerobic power output was made using the nomogram of Åstrand and Ryhming (1954).

Laboratory measurements

During the more detailed investigation of the cane cutters in the laboratory the measurements of body weight, height, blood Hb concentration, and skinfold thickness were repeated. From this latter measurement lean body mass (LBM) was estimated using the method of Durnin and Rahaman (1967). In addition, leg volume (LV) was determined from measurements of height and circumference of the thigh and calf and from direct measurements of water displacement after the method of Jones and Pearson (1969).

Exercise was performed on a stationary bicycle ergometer. During work the subjects inspired from a low-resistance CD4 Parkinson-Cowan gas meter and expired through an Otis-McKerrow valve into a short piece of wide tubing connected to a mixing chamber (Davies and Shirling, 1967). A continuous form of test was given: after an initial period of 3 minutes' warm up the workload was raised every 2 minutes by 200 kpm/min, the object being to cover the whole range of the subject's work capacity up to and including maximum effort. During the final minute at each workload, cardiac frequency was taken from a standard (Phillips) ECG recorder, respiratory frequency was counted, and inspired ventilation volume was recorded. At the same time samples of expired air were taken from the mixing chamber for subsequent analysis of CO_2 and O_2 content using a katharometer (Cambridge Instruments Ltd.) and a paramagnetic O_2 analyser (Servomex Ltd.) respectively. The meters were calibrated at frequent intervals using gases of known concentration.

From these data oxygen intake ($\dot{V}O_2$) was calculated and the regression equations of cardiac frequency (f_H) and minute ventilation ($\dot{V}_E$) on $\dot{V}O_2$, and of $\dot{V}O_2$ on f_H and workload (W) were computed for each subject individually and for the group as a whole. This enabled the results for exercise to be expressed in terms of a fixed $\dot{V}O_2$ of 1·5 l.min^{-1} (Cotes, Davies, Edholm, Healy, and Tanner, 1969) or a workload of 900 kpm·min^{-1}, and allowed the prediction of maximum aerobic power ($\dot{V}O_{2max}$).

Details of each individual's daily earnings and cane-cutting capacity were obtained from the records of the Company.

Results

Inter-subject variability in physique, productivity, and work capacity and seasonal differences in earnings

The data for this part of the investigation were taken at the beginning of the season. Measurements of age, weight, and height were made on the complete cane-cutting labour force of 585 men and on a subgroup of 285 of these workers. Determinations

of skinfold thickness, maximum calf and biceps circumference, blood haemoglobin, and physical working capacity (step test) were recorded. The aims of this part of the investigation were to establish whether (1) differences in body physique, fitness, and productivity did exist sufficient to justify a definitive study of the problem, and (2) the differences in cane-cutting performance in the fields were consistent from season to season. It will be seen (Table 1 and Fig. 2) that large differences do indeed exist for all parameters measured except blood haemoglobin but the figures for this parameter are not truly representative as all workers with a haemoglobin value less than 50% normal were eliminated before the investigation.

The mean weight, height, sum of four skinfold thicknesses, and biceps and calf circumferences are similar to those shown in Table 3 of the definitive study and show a 5 to 20% variation. The predicted values of maximum aerobic power ($\dot{V}O_{2\ max}$) which were calculated from the cardiac frequency responses to a simple exercise test (Åstrand and Ryhming, 1954) show a similar pattern of variation. The mean $\dot{V}O_{2\ max}$ of the 285 cane cutters is 2·92 ±1·23 l.min^{-1}, which is closely in agreement with the average value found previously for sedentary men (Davies, 1971; Davies, Mbelwa, Crockford, and Weiner, 1973) and young adults (but not athletes) in training (Davies and Knibbs, 1971).

The data for earnings of the previous season were difficult, if not impossible, to obtain on the complete sample due to the African custom of changing names at irregular intervals of time. Reliable details of the productivity of 32 men included in the present study are shown (Fig. 2). The values range from 40 to 170 kilotons of cane cut per season and clearly show that the productivity of individual workers is a consistent phenomenon, each man maintaining approximately his rank order from one season to the next. The correlation between the productivity figures of the 1971 and 1972 season is r = +0·73.

A subsample of 78 cane cutters out of the 585 employed by the sugar estate was studied in the laboratory towards the end of the cutting season (Fig. 1). They were divided into four groups (Table 2): group 1, high producers (mean output 3·51 kilotons/day); group 2, medium producers (mean output 3·04 kilotons/day); group 3, low producers (mean output 2·60 kilotons/day); and group 4, men over 35 years of age. The physical characteristics of the men are given in Table 3.

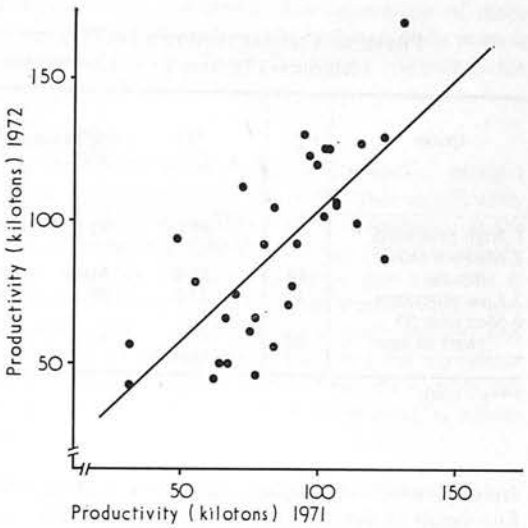

FIG. 2. Relationship of productivity in 1972 and 1971. The regression equation is of the form: y (1972) = 9·09 + 0·956x where y = productivity in 1972 season, and x = productivity in 1971 season (r = +0·73; P < 0·001).

Physiological responses to exercise

For a given workload of 900 kpm.min^{-1} the oxygen intake ($\dot{V}O_{2\ 900}$) is identical for all four groups studied (Table 4) and agrees closely with previous work from this and other laboratories (Davies, 1971; Åstrand, 1960). This is also true of the minute ventilation volume at the given $\dot{V}O_2$ of 1·5 l.min^{-1} ($\dot{V}_{E\ 1.5}$). The values range from 46 to 49

TABLE 2

ABSOLUTE AND DAILY PRODUCTIVITY (TOTAL CANE CUT AND CANE CUT PER DAY) AND ABSENTEEISM FROM CANE FIELDS

Group	*n*	*Absolute output (kilotons)*	*Relative output (ktons/day)*	*Days absent*
1 High producers ..	20	360·6***2,3,4	3·51***2,3,4	8·7***2,3,4
2 Medium producers	19	294·7	3·04***3	15·2***3
3 Low producers ..	19	252·2	2·60***4	21·1***4
4 Men over 35 years of age	20	287·4	2·95	14·7

*P < 0·05; **P < 0·01; ***P < 0·001

TABLE 3

PHYSICAL CHARACTERISTICS OF 78 SUBJECTS STUDIED DIVIDED INTO 4 GROUPS: HIGH (1), MEDIUM (2), AND LOW (3) PRODUCERS AND OLDER MEN (4): (MEAN ± SD)

Group	n	Age (yr)	Wt (kg)	Ht (cm)	Σsf (mm)	LBM (kg)	LV (l.)	Circumference	
								Biceps (cm)	Calf (cm)
1 High producers	20	25·6	62·9	167·5	25·2	55·7	12·6	27·4	34·9
2 Medium producers ..	19	25·9	61·4	166·1	25·6	54·2	12·4	27·8	34·8
3 Low producers	19	27·4	59·1	165·4	24·7	52·5	11·7	27·1	34·1
4 Men over 35 years of age..	20	38·8 ***1,2,3	64·4	169·8	24·2	57·3	12·8	27·7	35·0

***P <0·001

1.min^{-1}, which are higher than those recorded for Europeans at similar work levels (Davies, 1971) but in accord with previous findings for Africans (Ojikutu, Fox, Davies, and Davies, 1972). The differences in cardiac frequency ($f_{H\ 1·5}$) at a fixed $\dot{V}O_2$ are also small; the subjects in group 1 have a lower value than those in the other three groups but the difference is not significant. However, when the $f_H/\dot{V}O_2$ data are used to predict the $\dot{V}O_{2\ max}$, the high producers of group 1 have significantly (P < 0·001) higher aerobic power outputs than either group 3 or group 4 but not group 2 (Table 4). In relative terms (ml.min^{-1} kg^{-1} body weight) mean values of $\dot{V}O_{2\ max}$ range from 51 ml.min^{-1} kg^{-1} (group 1) to 43 ml.min^{-1} kg^{-1} (group 4), groups 2 and 3 having an intermediate value of approximately 48 ml.min^{-1} kg^{-1}.

Relationships of productivity to maximum aerobic power, physique, and body composition

For any given work output in the cane fields (kilotons of cane cut per season) the maximum aerobic power output as measured in the laboratory appears to be constant. The correlation between $\dot{V}O_{2\ max}$ and total output per season is r = +0·21, and the slope of the line does not differ significantly from zero. Thus in absolute terms one can conclude that the workers' productivity is independent of their $\dot{V}O_{2\ max}$. However, an important feature of the work in the cane fields is not only the output recorded in this way but also the number of days spent to produce a given output. In general, the high producers appear to spend less time (i.e., they work at a higher rate) to produce a given output and voluntarily absent themselves less from the cane fields (Table 2). The lower producer, on the other hand, has to work more days to achieve the same output and takes more unpaid rest days away from the fields. In Fig. 3 the data for $\dot{V}O_{2\ max}$ are plotted against daily productivity in kilotons per day. Although there remains a large scatter in individual values a definite positive association (r = +0·46; P < 0·001) emerges. In Table 5 the principal indices of body physique and composition have been related to productivity in both relative and absolute terms in the form of a correlation matrix for groups 1 and 3 (subjects younger than 35 years) and group 4 (subjects older than 35 years). Correlation coefficients

TABLE 4

SUBMAXIMAL RESPONSES TO EXERCISE AND PREDICTED MAXIMUM AEROBIC POWER (MEAN ± SD)

Group	n	$\dot{V}O_{2\ 900}$ (l.min^{-1})	$\dot{V}_{E\ 1·5}$ (l.min^{-1})	$f_{H\ 1·5}$ (beats.min^{-1})	Predicted $\dot{V}O_{2\ max}$	
					l.min^{-1}	ml.kg (body wt) min^{-1}
1 High producers	20	2·12	46·8	118	3·20***3,4	51·0***3,4
2 Medium producers ..	19	2·10	45·8	123	2·95	48·0
3 Low producers	19	2·09	48·6	124	2·80	47·3
4 Men over 35 years of age..	20	2·12	48·6	120	2·76	43·0

***P <0·001

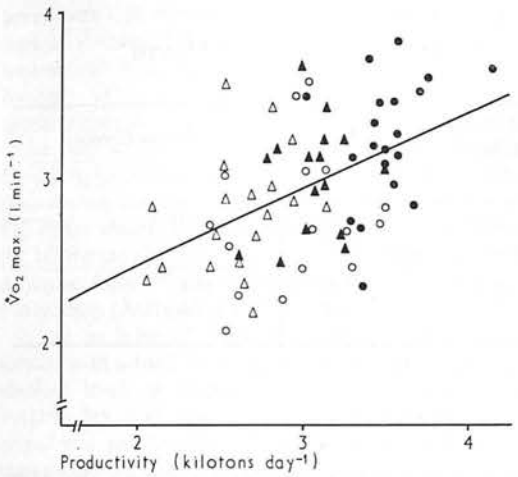

FIG. 3. Relationship of maximum aerobic power ($\dot{V}O_{2\,max}$) to relative productivity (cane cut in kilotons per day). The regression is of the form: $y=0{\cdot}449x+1{\cdot}573$ where $y=\dot{V}O_{2\,max}$, and x=relative productivity ($r=+0{\cdot}46$; $P<0{\cdot}001$): ● group 1; ▲ group 2; △ group 3; ○ group 4.

at or below the 5% level of significance have been shown. In terms of total productivity (output/season) no association is shown with the various indices of body composition for either the younger or older subjects. However, for daily productivity (output/day) significant relationships are shown in the younger subjects for body weight (0·36), lean body mass (0·36), and limb volume (0·34), but in older subjects these relationships disappear. In groups 1 to 4 combined, the association of daily productivity with both weight and lean body mass is reduced to $r=+0{\cdot}24$ and disappears completely for limb volume.

Absenteeism

It is clear from Table 2 that the workers in group 1 absent themselves from the cane fields significantly less than those in groups 2 to 4. Attendance at the fields is voluntary and if a worker absents himself, other than for sickness, he loses pay and this is recorded on his personal file. It is in the worker's own interest to attend the fields daily except Sunday and it is therefore of interest to find that the maximum aerobic power shows a very small ($r=-0{\cdot}32$) but significant ($P<0{\cdot}001$) negative relationship to absenteeism from the cane fields.

Effects of cane-cutting season on body weight and composition

Since measurements of the major indices of body size and composition were taken before and after the 1972 cane-cutting season, it is possible to assess the effects of six months' hard physical work on these parameters (Table 6). The cane cutters were responsible for finding their own food except for morning tea and maize porridge which was supplied by the Estate. Under these conditions there is a small but significant loss of body weight ($P<0{\cdot}05$) and lean body mass ($P<0{\cdot}01$) which is accompanied by an increase ($P<0{\cdot}001$) in biceps and calf girth.

Discussion

In the developed countries of the world, although manual labour is still important in some industrial

TABLE 5

CORRELATION MATRIX FOR AGE, WEIGHT, HEIGHT, SUM OF 4 SKINFOLD THICKNESSES, LEAN BODY MASS, LEG VOLUME, MAXIMUM (BICEPS AND CALF) CIRCUMFERENCES, PRODUCTIVITY (ABSOLUTE AND RELATIVE) AND MAXIMUM AEROBIC POWER ($\dot{V}O_{2max}$) FOR GROUPS 1 TO 3 COMBINED AND GROUP 4

Group 4 / Groups 1-3	*Age*	*Wt*	*Ht*	*Σsf*	*LBM*	*LV*	*Biceps*	*Calf*	*Output (absolute)*	*Output (relative)*
Age										
Wt			0·56		0·98	0·94	0·85	0·95		
Ht		0·60			0·60	0·57		0·56		
Σsf		0·47	0·34				0·48			
LBM		0·98	0·58	0·28		0·94	0·80	0·94		
LV		0·87	0·59	0·36	0·87		0·74	0·91		
Biceps		0·73	0·34	0·43	0·69	0·63		0·82		
Calf		0·79	0·37	0·29	0·80	0·72	0·59			
Output (absolute)										
Output (relative)		0·36			0·36	0·34			0·43	

TABLE 6

PHYSICAL CHARACTERISTICS OF 37 CANE CUTTERS BEFORE AND AFTER A 6-MONTH CANE-CUTTING SEASON (MEAN ± SD)

	Wt (kg)	*Ht (cm)*	*Σsf (mm)*	*LBM (kg)*	*Circumference*	
					Biceps (cm)	*Calf (cm)*
Before n=37	63·5* ±6·8	167·4 ±5·6	22·5* ±3·3	57·06** ±5·91	26·3*** ±1·6	34·0*** ±2·1
After n=37	62·1 ±7·4	167·2 ±5·8	24·4 ±5·5	55·51 ±6·07	27·6 ±1·6	35·0 ±2·3

*P <0·05; **P <0·01; ***P <0·001

spheres there has been a rapid move towards mechanization and automation over the past 20 years so that it may no longer be necessary for large sections of the community to exert themselves physically for prolonged periods in order to earn a living. In developing countries, however, conditions are very different. The vast majority of the population still depend mainly on subsistence farming to meet their daily requirements for food and shelter, and agriculture forms the governments' basic policy to meet the challenge of economic growth. In Tanzania, where the present study was undertaken, the move towards mechanization is slow and the need for human muscle power in various forms of rural and urban occupation remains. In this situation, it is of vital importance that occupational and industrial physiologists should strive towards a fuller understanding of the factors which may contribute to higher work output and increased efficiency so that once identified they can be incorporated into procedures for training and selecting personnel in order to raise the general productivity of the nation.

In the present study, which was designed to investigate the relationship between the physiological responses to exercise as measured in the laboratory to actual work output during a given task of cane cutting, one factor does clearly emerge: that there is a significant association between maximum aerobic power output ($\dot{V}O_{2\ max}$) and the daily rate of working in the cane fields. High producers (group 1) possessed ~20% advantage in $\dot{V}O_{2\ max}$ over low producers (group 3), which was exactly paralleled by the difference in work output in the cane fields between the two groups (Table 2). Further, the decline in aerobic power output of the older subjects in group 4 is reflected in a reduced work output in the field. These results are clearly at variance with those of Wyndham *et al.* (1964), who showed no such relationship to exist in African Bantu workers employed in the gold mines. However, it is difficult to compare the two sets of data exactly because of the very different conditions in which they were collected. We agree with the importance they attach to such factors as skill, motivation, and leadership in contributing to given work output. In most human activities there is probably always a strong interplay between psychological and physiological factors which serve to confound the investigator. In the present investigation it must be stressed that the relationship we have found (Fig. 3) accounts for only 25% of the variance of productivity and clearly many other factors are involved. They may include, as well as those cited above, experience, attitudes, mood, the use of rest pauses, and state of acclimatization. Nevertheless our data suggest that the $\dot{V}O_{2\ max}$ does provide a useful guide on a group basis to productivity and probably sets the upper limits of potential performance for the individual.

The maximum aerobic power is related not only to the rate of work in the cane field in the present study but also to absenteeism, a finding which is in agreement with the work of Lindén (1969). We found this aspect of the investigation difficult to study quantitatively. Our attempts to elucidate from the workers why they voluntarily absented themselves from the fields yielded many inconsistencies. However, one factor did emerge: the cane cutters often expressed the view that they simply did not feel up to the work and therefore, despite the loss of wages, would decide to spend the day in the compound. They did not feel sick or unwell in the accepted sense but were unable to face a heavy day's work in the heat. It may well be that the $\dot{V}O_{2\ max}$ not only sets the upper limit of potential performance but confers on those with a high value the ability to work at high rates for long periods of time. Thus they are able to work within their physiological capacity and are not forced to greater efforts than they are capable of making. They thereby avoid the cumulative day to day effects of fatigue, which leaves them free to pursue their jobs consistently throughout the cane-cutting season. Certainly the present data are in

accord with this view and find support in the earlier work of Åstrand (1967). In her study of building workers she was able to establish a relationship between $\dot{V}O_{2\,max}$ and occupational workload spontaneously chosen by the individual if given a free choice. The workers in her study varied in $\dot{V}O_{2\,max}$ from 2·19 to 3·63 l.min^{-1} (Table 4) but habitually chose a work rate corresponding to ~40% $\dot{V}O_{2\,max}$. This agrees closely with the rate of work she found in the laboratory beyond which both objective and subjective signs of stress occurred if prolonged for 6 to 8 hours (Åstrand, 1960).

It can be inferred from her findings that those subjects with a high $\dot{V}O_{2\,max}$ would achieve a greater absolute level of energy expenditure during the working day and presumably attain greater work output and productivity. She also showed that the older workers' $\dot{V}O_{2\,max}$ declined with age (Table 4) but the difference between the level of maximum aerobic power output of her old workers and ours should be noted. The older African cane cutters in the present study possess exceptional maximal aerobic power levels compared with Europeans of the same age (Davies, 1971). They show no increase in either body weight or body fat (Table 3), and their $\dot{V}O_{2\,max}$ values are similar to those for young healthy men of European descent (Cotes *et al.*, 1969; Davies, 1971).

Practical implications

To be of practical use the factors which underlie productivity must have some predictive value. Clearly, as already mentioned, on an individual basis the predictive power of $\dot{V}O_{2\,max}$ to estimate productivity is useless. For a given $\dot{V}O_{2\,max}$ value of, for example, 3 l.min^{-1} the confidence limits are such that we might expect his productivity in the cane fields to lie anywhere between 2·29 and 3·85 kilotons/day. This covers the whole range of work outputs measured, from the highest in group 1 to the lowest in group 3. However, on a group basis, the picture is different. It will be seen (Table 5) that daily work output as well as being related to $\dot{V}O_{2\,max}$ is also associated with measurements of body size and composition. This may give some support to the procedure adopted in some industries (e.g., Chamber of Mines, South Africa) which entails hard physical work in order to eliminate those subjects with low body weight, LBM, or LV. But the data given in Table 3 would indicate that such measurements are not decisive in assessing potential productivity, and the power output to weight or LBM ratio may be a more important factor in cane cutting. In the cane-cutting industry selection is essential due to the overwhelming number of applications for the limited jobs available. It might therefore be useful at the time of recruitment to include a simple measure of work capacity together with body weight and LBM determinations at the time of the general medical examination, which should also include screening tests for parasitic infection and anaemia. Certainly it is our experience (Davies, Chukweumeka, and Van Haaran, 1972) that subjects with Hb levels less than 9 g/100 ml should be excluded. The work capacity test we gave in the laboratory is too sophisticated and time-consuming for practical purposes. The results of a simple step test (Table 1) may be more relevant. The test was extremely crude and again the individual values had little meaning but correlated well ($r = +0{\cdot}57$) with the $\dot{V}O_{2\,max}$ measured in the laboratory, in 32 cane cutters for whom data were available. The test is short, simple, requires a minimum of equipment, and could readily be incorporated into selection procedures.

Finally, the effects of a season's hard physical work on the body composition of the cane cutter are minimal but significant (Table 6) and they are quite contrary to studies which have been done on well-fed Europeans undergoing training (Carter and Phillips, 1969; Ribisi, 1969; Pollock *et al.*, 1971). In the present investigation, body weight declined by 1·4 kg ($P < 0{\cdot}05$) but this was surprisingly accompanied by an increase in fat ($P < 0{\cdot}05$), a decrease in LBM ($P < 0{\cdot}01$), and an increase of biceps and calf circumferences ($P < 0{\cdot}001$). These changes may be related to the effects of hard physical work on subjects who were existing on a subsistence diet rich in carbohydrate (mainly in the form of maize) and poor in animal protein, and fully underlines the need for further research work.

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IRON-DEFICIENCY ANAEMIA: ITS EFFECT ON MAXIMUM AEROBIC POWER AND RESPONSES TO EXERCISE IN AFRICAN MALES AGED 17–40 YEARS

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SUMMARY

1. The physiological responses to exercise of two groups of industrial workers with moderate and severe anaemia were measured and compared with a group of matched controls, working and living under similar conditions. Exercise was performed on a stationary bicycle ergometer at five consecutive work loads. During the final minute of each exercise periods, oxygen intake, minute ventilation volume, and cardiac and respiratory frequencies were measured. On a separate occasion, blood haemoglobin concentration was measured at rest and cardiac output and blood and plasma volumes were estimated while subjects exercised at a fixed percentage of the maximum aerobic power for 6 min.

2. Oxygen intake for a given work load and ventilation rate was similar in all subjects studied but exercise cardiac output was elevated in the anaemic group. The increased cardiac output was due to an increased heart rate, the stroke volume being similar in the anaemic and control subjects. There was a marked decrement (~34%) in maximum aerobic power and some evidence of a raised plasma volume in the severely anaemic group.

3. These results suggest that anaemia impairs performance during moderate and near maximum exercise. There is an associated rise of cardiac output and plasma volume.

Key words: anaemia, oxygen consumption, exercise, cardiac output.

There have been few studies of the physiological responses to exercise of anaemic subjects and the evidence from these is conflicting. Beutler, Larsh & Tanzi (1960), for instance, report that during exercise the transport of oxygen to the working tissues is unaffected by a decrease in haemoglobin concentration, whereas Sproule, Mitchell & Miller (1960) show clearly that severe anaemia produced a large decrement (>50%) of maximum aerobic power ($\dot{V}O_{2,\,max.}$).

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These authors also reported that the anaemic subjects are able to exercise at similar levels of maximum work (despite their reduced $\dot{V}O_{2,\,max.}$) as the controls without signs of symptomatic stress. This may mean that severe anaemia results in the greater utilization of anaerobic sources of energy during work. However, the authors presented no evidence on this point, and their work has not been confirmed by the recent studies of Cotes, Dabbs, Elwood, Hall, McDonald & Saunders (1972). They, like Beutler and his co-workers (1960), found no evidence that moderate anaemia (Hb = 8·6 g/100 ml) either impaired work performance or was associated with compensatory cardiovascular adjustments.

In the present investigation we have attempted to resolve these differences in experimental observations, by comparing the cardiovascular responses and exercise tolerance of two groups of industrial workers with moderate (Hb = 8–10 g/100 ml) and severe (Hb < 8 g/100 ml) anaemia with matched normal controls (Hb > 13 g/100 ml) working and living under similar conditions.

MATERIALS AND METHODS

The subjects were African industrial workers living and working in Dar es Salaam. Their physical characteristics together with circulating haemoglobin levels are given in Table 1. The subjects were identified during an epidemiological survey of industrial anaemia. The

TABLE 1. The age, height, weight, blood haemoglobin concentration (Hb) and blood (V_b) and plasma (V_p) volumes of the controls and anaemic subjects given as means ± SD. Measurements of V_p and V_b were made on five subjects in each group.

Group	Age (years)	Height (cm)	Weight (kg)	Hb (g/100 ml)	V_p (litres)	V_b (litres)
Controls	27·1	165·6	54.7	14·5*** II,III	2·3*** III	3·9
I (n = 16)	±7·1	±4·3	±5·4	±1·5	±0·3	±0·4
Moderately anaemic	23·7	164·0	50·9	9·2*** III	2·4	3·5
II (n = 8)	±9·7	±7·2	±8·4	±0·8	±0·8	±1·3
Severely anaemic	22·0	163·7	54·4	6·7	3·0	4·0
III (n = 9)	±6·2	±2·8	±5·4	±1·1	±0·4	±0·2

*** Value significantly different (P < 0·001) from the group shown by the roman numerals.

severe anaemic group comprised all those found with a haemoglobin concentration of less than 8 g/100 ml. Those with moderate anaemia (Hb = 8–10 g/100 ml) were randomly selected from a group of 200 workers. Each subject in groups II and III (Table 1) was matched approximately with a normal control (Hb > 13 g/100 ml) on the basis of age, weight, height, job and place of work, the aim being as far as possible to use work-mates performing identical tasks in the same factory. All subjects were examined clinically and a chest X-ray and lung-function tests were performed. If they were free of symptoms (except those arising from the anaemia), the nature of the experiment was explained to them and, if they agreed, they were included in the study.

The subjects attended the laboratory on a single occasion. During the preliminary session they were allowed to become familiar with the procedures (Davies, Tuxworth & Young, 1970). Following this period the subjects were then required to exercise for five consecutive 3 min

periods at 180, 360, 540, 720 and 900 kpm/min. During the final minute of each period oxygen intake ($\dot{V}O_2$), minute ventilation ($\dot{V}_E$), cardiac ($\dot{f}_H$) and respiratory ($\dot{f}_R$) frequencies were measured. Finally, after a 1 h rest-period haemoglobin concentration was measured at rest and cardiac output ($\dot{Q}$), blood (V_b) and plasma (V_p) volumes, $\dot{V}O_2$ and $\dot{f}_H$ determined with the subjects exercising at a fixed ($\sim 60\%$) percentage of their maximum aerobic power as judged from their previous work performance.

Minute ventilation and $\dot{V}O_2$ was measured using a standard open circuit technique previously described (Davies, 1968). The subjects breathed through a low-resistance Otis–McKerrow valve and expired air, collected into Douglas bags, was analysed for O_2 and CO_2 content using a paramagnetic Servomex O_2 analyser and a katherometer. These instruments were calibrated at frequent intervals using standard gas mixtures previously analysed by the Lloyd–Haldane chemical method. The accuracy of these techniques has been previously reported (Davies & Shirling, 1967). Cardiac frequency was determined from a continuous recording of the electrocardiogram. For measurement of blood and plasma volumes a 2% solution of Evans Blue containing approximately 3 mg of dye was injected into an ante-cubital vein of the left arm and samples were withdrawn from the right arm after 5, 10 and 15 min. A straight line was fitted by eye to the semi-logarithmic plot of dye concentration against time and the theoretical concentration of dye at zero time was estimated by extrapolation. Blood and plasma volumes were then derived in the standard way. The blood haemoglobin concentration was determined by the cyanmethaemoglobin method using an EEL photometer; duplicate measurements were required to agree within $\pm 0{\cdot}5$ g/100 ml. Cardiac output was estimated by the indirect Fick technique using an ear oximeter. Oxygen intake was calculated from ventilatory and expired gas data and individual regression lines were fitted by the method of least squares to plots of minute ventilation volume and cardiac frequency on $\dot{V}O_2$, and to $\dot{V}O_2$ on work load and cardiac frequency. From these data, $\dot{V}O_2$ was expressed at a fixed work load of 900 kpm/min ($\dot{V}O_{2,\,900}$); and maximum aerobic power ($\dot{V}O_{2,\,max.}$) was predicted from an extrapolation of the fitted $\dot{f}_H/\dot{V}O_2$ line to a cardiac frequency of 195 beats/min. Minute ventilation volume and cardiac frequency were expressed at a $\dot{V}O_2$ of 1·5 litres/min ($\dot{V}_{1\cdot5}$ and $\dot{f}_{H,\,1\cdot5}$) respectively (see Cotes, Davies, Edholm, Healey & Tanner, 1969).

RESULTS

The blood and plasma volumes of the anaemic subjects and their controls are given in Table 1. The responses to submaximal exercise are summarized in Table 2 and Figs. 1–3. The severely (mean Hb = 6·7 g/100 ml), but not the moderately (mean Hb = 9·2 g/100 ml) anaemic group, have a significantly ($P<0{\cdot}001$) larger plasma volume than their controls. However, this does not give rise to an elevated blood volume but merely compensates for the lower packed cell volume of the anaemic subjects.

For a given $\dot{V}O_2$ the severely anaemic subjects exercise with a higher heart rate ($P<0{\cdot}001$) than the controls; this is also true of the moderately anaemic group but the magnitude of the response is reduced (Table 2). Similarly, minute ventilation at a given $\dot{V}O_2$ appears slightly higher in the severely anaemic group, but larger inter-subject variability precludes these differences reaching levels of significance. For a given work load the $\dot{V}O_2$ is almost identical for the three groups. The mean overall mechanical efficiency for the subjects of this study is close to 23%; a value similar to that found for Europeans during work on a bicycle ergometer.

TABLE 2. The responses to exercise expressed in terms of an oxygen intake ($\dot{V}O_2$) at a work load of 900 kpm/min ($\dot{V}O_{2,\,900}$), minute ventilation volume ($\dot{V}_{E,\,1.5}$) and cardiac frequency ($\dot{f}_{H,\,1.5}$) at a $\dot{V}O_2$ of 1·5 litres/min and predicted maximum aerobic power ($\dot{V}O_{2,\,max.}$) given as means ± SD.

Group	$\dot{V}O_{2,\,900}$ (litres/min)	$\dot{V}_{E,1.5}$ (litres/min)	$\dot{f}_{H,\,1.5}$ (beats/min)	$\dot{V}O_{2,\,max,}$ (litres/min)
Controls	2·01	49·4	127*** II,III	2·88*** III
I (n = 16)	±0·12	±13·5	±9	±0·46 **II
Moderately anaemic	2·10	49·3	160	2·20
II (n = 7)	±0·20	±6·3	±18	±0·42
Severely anaemic	2·03	52·6	170	1·90
III (n = 10)	±0·18	±10·6	±18	±0·37

** and ***, values significantly different (** $P < 0{\cdot}01$; *** $P < 0{\cdot}001$) from the groups, shown by the roman numerals.

The higher heart rate found in the anaemic groups is accompanied by an elevated cardiac output. Thus at a given $\dot{V}O_2$, stroke volume (SV) is essentially similar in the three groups (Fig. 2). The major difference in circulatory response between the groups is in the arterio-venous O_2 difference. For a given $\dot{V}O_2$ this is lower in the anaemic groups than in their controls, the difference becoming particularly noticeable at higher levels of work (Fig. 3).

Predicted maximum aerobic power

The predicted maximum aerobic power ($\dot{V}O_{2,\,max.}$) values are given in Table 2. In absolute

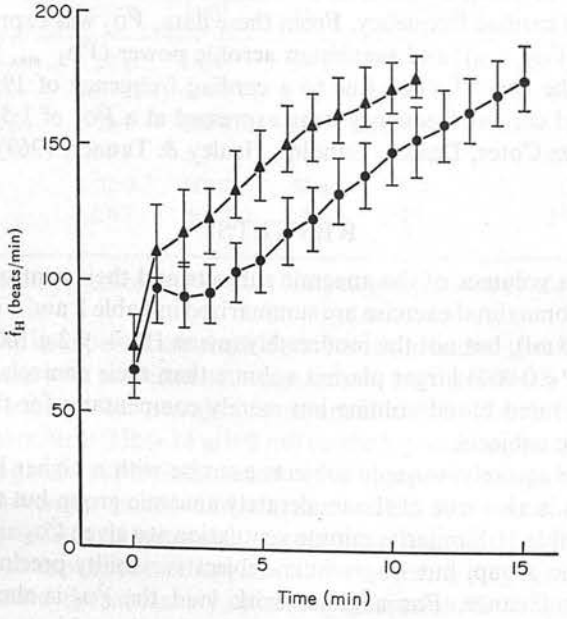

FIG. 1. Relationship between cardiac frequency ($\dot{f}_H$) and time for anaemic (▲) and normal (●) subjects (means ± SD) during the continuous submaximal test.

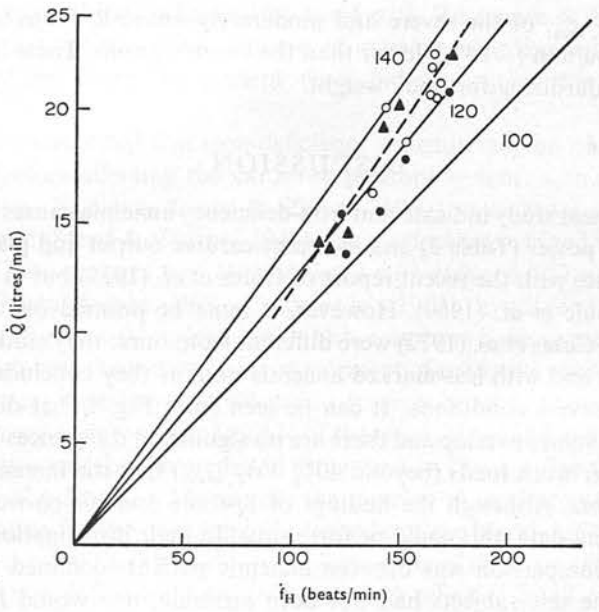

FIG. 2. Relationship of cardiac output ($\dot{Q}$) to cardiac frequency ($\dot{f}_H$). The isopleths are given for stroke volumes in ml. ○, Group I (controls); ●, group II (moderately anaemic); ▲, group III (severely anaemic). – – – –, mean regression line.

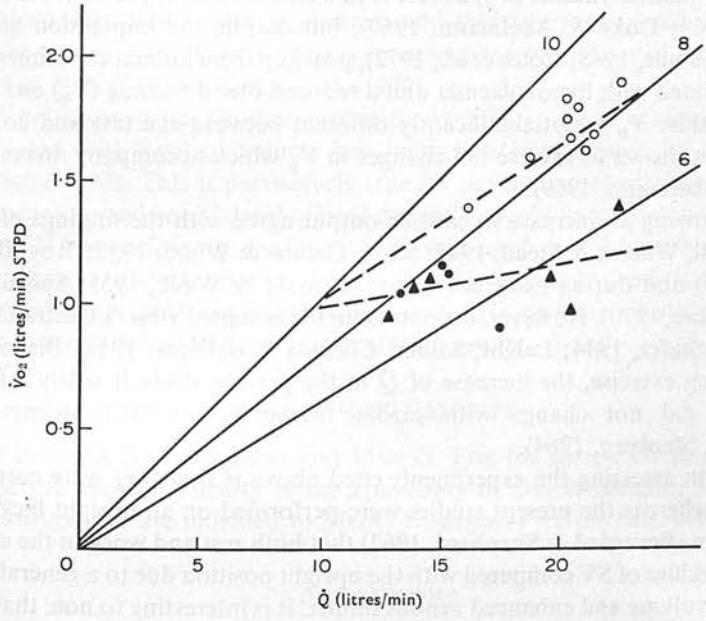

FIG. 3. Relationship of oxygen uptake ($\dot{V}O_2$) to cardiac output ($\dot{Q}$). The isopleths are shown for arterio-venous O_2 difference in vol. %. Symbols are as in Fig. 3.

terms the mean $\dot{V}O_{2,\,max.}$ of the severe and moderately anaemic subjects are 0·98 litre/min (~34%) and 0·68 litre/min (~24%) lower than the control group. These large differences are not removed by standardizing for body weight.

DISCUSSION

The results of the present study indicate that iron-deficiency anaemia causes a marked reduction in maximum aerobic power (Table 2) and increases cardiac output and plasma volume. These findings are at variance with the recent report of Cotes *et al.* (1972) but in agreement with the earlier work of Sproule *et al.* (1960). However, it must be pointed out that the conditions in the experiments of Cotes *et al.* (1972) were different from ours: they studied women working at lighter work loads and with less-marked anaemia and, as they conclude, it is dangerous to extrapolate to more severe conditions. It can be seen from Fig. 1 that during light work ($\dot{f}_H$ 110 beats/min) the $\dot{f}_H$ values overlap and there are no significant differences between the groups. It is only at the higher work loads (beyond 40% $\dot{V}O_{2,\,max.}$) that the increase in heart rate and cardiac output appears. Although the findings of Sproule and his co-workers (1960) are in accord with the present data, this could be fortuitous. In their investigation, matched controls were not used; the comparison was between anaemic patients confined to bed and healthy young subjects. If the test subjects had not been anaemic, one would have expected large differences in maximum aerobic power between these two groups of subjects (Saltin, Blomquist, Mitchell, Johnson, Wildenthal & Chapman, 1968). Our subjects were matched for age, height, weight (see Table 1) and daily (but not recreational) habitual activity. The test subjects and the controls were work-mates from the same factory living and working under identical conditions.

An increase of plasma volume (V_p) at rest is in agreement with previous studies (Backman, 1961; Cropp, 1969; Duke & Abelmann, 1969), but despite the impression gained by some (Blumgart & Altschule, 1948; Cotes *et al.*, 1972), possibly from animal experiments, that severe anaemia is associated with hypovolaemia and a reduced blood volume (V_b) our results in man are contrary to this: V_b is not significantly different between the test and control subjects. Therapy has been shown to reverse the changes in V_p which accompany anaemia (Backman, 1961; Duke & Abelmann, 1969).

Our results showing an increase in cardiac output agree with the findings of others at rest (Brannon, Merrill, Warren & Stead, 1945; Starr, Collins & Wood, 1933; Roy, Bhatia, Mathur & Virmani, 1963) and during exercise (Bishop, Donald & Wade, 1955; Sproule *et al.*, 1960; Anderson & Barkve, 1970). However, in contrast to the accepted view (Liljestrand & Stenstrom, 1925; Sharpey-Schafer, 1944; Leight, Saider, Clifford & Hellems, 1951; Bishop *et al.*, 1955) at rest and during exercise, the increase of $\dot{Q}$ in the present study is solely a function of $\dot{f}_H$. Stroke volume did not change with cardiac frequencies >110 beats/min (cf. Åstrand, Cuddy, Saltin & Stenberg, 1964).

The difficulty in assessing the experiments cited above is that they were carried out in the supine position whereas the present studies were performed on an upright bicycle ergometer. It has been shown (Bevegård & Shepherd, 1967) that both rest and work in the supine position produces larger values of SV compared with the upright position due to a general redistribution of central blood volume and enhanced venous return. It is interesting to note that Sproule *et al.* (1960) found that although anaemic subjects had a larger SV at rest than controls the changes in exercise were only such as to make the two groups comparable. We believe that the circulatory

system of the anaemic subject behaves in accord with Rushmore & Smith's original view for normal adults: that the major determinant of increase incardiac output is a rise in $\dot{f}_H$, SV remaining constant, and from the present data, independent of the level of circulating haemoglobin.

Many authors have suggested that iron-deficiency anaemia may be wholly compensated for by one or several factors affecting the cardio-respiratory system: a shift to the right of the O_2-dissociation curve (Rodman, Close & Purcell, 1960), a decrease in venous saturation and vascular resistance (Stead & Warren, 1947) and a reduction in blood viscosity (Richardson & Guyton, 1959). These changes may be associated with reduced diffusion capacity (Guleria, Pande, Markose, Gupta & Jain, 1971; Cotes *et al.*, 1972), hypoxaemia and an increase in alveolar–arterial O_2 gradient (Housley, 1967). Other authors have suggested from the results of animal experiments that there may be an impairment of oxidative enzymes at the tissue level in anaemia. We have no experimental evidence to confirm or refute these claims but the differences in the cardiac output data (Figs. 2 and 3) of the test subjects and controls at near maximal exercise adequately account for the predicted differences in $\dot{V}O_{2,\,max.}$ for the two groups shown in Table 2. Further, at a $\dot{V}O_2$ of 1·5 litres/min the increase in cardiac output of 8 litres/min is sufficient to compensate for the decreased O_2 capacity of the blood due to a circulating blood Hb level of approx. 6 g/100 ml. Thus although all or some of the above cited mechanisms may operate in exercise, our data would suggest that their effect is small and less important in anaemia than the compensatory tachycardia and increased cardiac output of work.

Despite this increase in cardiac rate and output, we find no support in the present study for the view of Sproule *et al.* (1960) that the anaerobic component of work is increased in anaemic subjects. For a given work load of 900 kpm/min, the $\dot{V}O_2$ of the test and control subjects is identical (Table 2). All the subjects in the investigation pedalled with similar mechanical efficiency independently of the level of anaemia and there were no significant differences in $\dot{V}_{1·5}$ between the groups. This latter finding is in agreement with those of Richards & Strauss (1928), Guleria *et al.* (1971) and Cotes *et al.* (1972).

The marked reduction of maximum aerobic power ($\dot{V}O_{2,\,max.}$) in anaemic subjects of 1·0 litre/min compared with control values, has important implications for the clinical and industrial medicine fields. This is particularly true for developing countries where anaemia is endemic and economic and social development depends largely on subsistence agriculture and physical labour. If iron-deficiency anaemia is left untreated it will have a serious effect on human work-output and performance. It should also be emphasized that if exercise tests are used in clinical medicine in situations where anaemia is endemic or suspected, then there is a need for standardization and care in interpreting results.

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RDIAC FREQUENCY DURING SUBMAXIMAL EXERCISE IN YOUNG ADULTS; RELATION TO LEAN BODY MASS, TOTAL BODY POTASSIUM AND AMOUNT OF LEG MUSCLE.

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The cardiac frequency and consumption of oxygen during submaximal exercise, lean body mass, total body potassium, thigh muscle width and an index of thigh muscle volume have been obtained for seventy-nine healthy men and women, ages 18 to 38 yrs, who were not in athletic training but accustomed to taking some exercise. For these subjects, at any specified submaximal rate of consumption of oxygen, the cardiac frequency is negatively correlated with the indices of body muscle which are themselves inter-correlated ($r > 0{\cdot}89$). The latter relationships provide a basis for prediction of total body potassium (coefficient of variation 6·1 %).

The relationship of exercise cardiac frequency to body muscle is similar in both men and women; it provides a better description of frequency than one based on consumption of oxygen alone (coefficient of variation respectively 12% and 18%). Cardiac frequency is independently related to the level of habitual activity of the subjects. The implications of these findings are discussed.

ing data for young adults not in athletic training this paper examines the lationship of the cardiac frequency during submaximal exercise to indices body muscle and shows that the two are related when allowance is also made r the consumption of oxygen. The basis for this relationship is set out below; e implications are considered in the discussion.

The cardiac frequency and the cardiac output during submaximal exercise e linearly related to the consumption of oxygen; but whereas the former lationship is known to vary between subjects, the latter is relatively constant evregard, Holmgren and Jonsson, 1963; Reeves, Groves, Blout and Filley, 61]. The linearity of the two relationships implies that for an individual bject the cardiac stroke output is relatively independent of the rate of work; is is the case for exercise in both a supine and an upright posture, though the latter the stroke output is larger during mild exercise than it is at rest evregard *et al.*, 1963]. The stroke output is the resultant of a number of factors which one must be the muscularity of the heart. Since the cardiac output the product of the stroke output and the frequency, the frequency at any vel of cardiac output should also be related to the amount of muscle. The esent study explores this relationship in groups of subjects by measuring

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oxygen consumption rather than cardiac output; the two are highly correlated, and oxygen consumption is much more convenient to measure on members of the general population.

The muscularity of the heart in healthy subjects may be represented by its overall size but no allowance is then made for differences in the respective dimensions of the walls and the cavities of the heart. However, the size of the heart is correlated with indices of body muscle, including the lean body mass and the width of the muscle of the upper thigh [$r \simeq 0{\cdot}6$; Cotes, Dabbs, Daley, Hall, Johnson, Reed and Saunders, 1971]. Other indices which reflect the quantity of the body muscle include the total body potassium [Brozek, 1965] and the volume of muscle in the leg [Jones and Pearson, 1969]. Thus we have looked for and in the event have found an association between the exercise cardiac frequency and these indices of body muscle. This finding is consistent with the view that in healthy young subjects the organs which contribute to the capacity for exercise are in general matched for size; their characteristics may be revealed by studies at rest or during submaximal exercise and may be used as a basis for analysis of the effects of other factors.

Subjects and Methods

Group A comprised thirty-nine young adult factory workers of both sexes whose performance during maximal exercise has been reported previously [Cotes, Davies, Edholm, Healy and Tanner, 1969]. The relationships of cardiac frequency to the indices of body muscle for these subjects were then applied to data for group B, forty trainee teachers of the same age group, of the women were taking general courses which included some physical recreation and the men were students of physical recreation [Jones, Cotes, Knibbs and Burkinshaw, 1970]. Details of the subjects and a summary of the physiological data which provide the basis for the present analysis are given in Table I; the habitual levels of activity are considered in the discussion.

The exercise was performed on a cycle ergometer. In group A all the subjects exercised at 450 k.p.m. min^{-1} (73·5 W) and a higher work rate which was for men 900 k.p.m. min^{-1} (147 W) and for women 720 or in two instances 540 k.p.m. min^{-1} (118 and 88 W respectively). The subjects in group B performed eight periods of work; the lowest work rate was 300 k.p.m. min^{-1} (29 W) and the other rates spanned a range which was adjusted for each subject according to his or her estimated capacity for exercise. These measurements were spread over 2 days. In both studies cardiac frequency and uptake of oxygen were obtained in the 5th and 6th min of exercise. The cardiac frequency was measured by electrocardiography and the oxygen uptake by the Douglas bag method using a low resistance valve box, tubing of internal diameter 2·8 cm and calibrated dry gas meter and physical gas analysers. The bags were suspended vertically to minimize their resistance. From these data the cardiac frequencies at oxygen uptakes of 1·0 and 1·5 l. min^{-1} were obtained subsequently by interpolation. Using a similar exercise procedure the coefficient of variation of a single determination in unexperienced subjects was 3·1% [Edwards, Miller, Hearn and Cotes, 1972]. The lean body mass was obtained indirectly from body fat which was estimated by the method of Durnin and Rahaman [1967] from the sum of four skinfold thicknesses measured with Harpenden skin calipers over the biceps and triceps muscles and in the subscapular and suprailiac regions. Durnin and Rahaman calibrated the method using measurements of body density, when the coefficient of variation about the regression line was 3·5%. The thigh muscle width was measured below the gluteal fold with the subject erect. The level adopted was one third of the

TABLE I. *Details of the subjects: mean values and ranges*

		Series A		Series B		Overall
		Men	Women	Men	Women	mean
Number		22	17	28	12	79
Age	(a)	24.9 (18·9–27·6)	23·7 (20·4–27·2)	26·8 (18·6–38·2)	18·7 (18·0–19·6)	24·4
Height	(m)	1·75(1·62–1·88)	1·61(1·50–1·70)	1·73(1·64–1·88)	1·65(1·60–1·73)	1·70
Weight	(kg)	72·4 (56·6–106)	55·7 (47·3–67·9)	70·0 (49·7–86·0)	60·8 (51·7–76·5)	66·2
Lean body mass	(kg)	62·4 (50·9–91·0)	40·5 (35.5–48·3)	60·3 (45·6–72·4)	43·1 (36·0–52·4)	54·0
Thigh muscle width	(cm)	13·4 (11·5–17·8)	10·6 (8·6–12·0)	13·60(11·0–15·5)	10·99(9·4–11·9)	12·5
Total body potassium	(mole)	3·83(2·91–6·39)	2·32(1·97–2·77)	3·85(2·83–4·57)	2·51(2·09–2·83)	3·31
Thigh muscle volume	(l.)	10·3 (6·6–16·4)	4·9 (3·8–6·5)	9·5 (6·6–13·0)	6·0 (4·8–7·7)	8·2
Cardiac frequency						
at $\dot{V}o_2$ 1·0	(min^{-1})	101(82–130)	140(103–173)	102(78–130)	130(120–145)	114
at $\dot{V}o_2$ 1·5	(min^{-1})	122(98–152)	169(131–192)	122(98–159)	164(151–181)	139

$\dot{V}o_2$ = consumption of oxygen, (l. min^{-1}.)

subischial height above the lower femoral condyles and the muscle width was obtained by radiography with the subject wearing a protective garment [Jones, 1971]. The total radiation dose to the skin was less than 50 mrad. Further details of the methods and results are available elsewhere [Cotes *et al.*, 1969].

The total body potassium was measured with a whole-body radiation counter; this was calibrated in a previous study in which ^{42}K was given as an internal standard to sixty-two subjects. The coefficient of variation of the estimates was about 4% and the reproducibility 1·5% [Burkinshaw, 1967]. The volume of muscle in the leg was obtained as muscle plus bone volume using the method of Jones and Pearson [1969]. This treats the leg as six segments, each having the configuration of a truncated cone. The lengths and circumferences of the segments are obtained using a digital reading anthropometer and a steel measuring tape and the thickness of the subcutaneous fat is obtained from measurements of skinfold thickness using calipers. The method was validated by measurements of volume made by water displacement and of thickness of the skin and subcutaneous tissue made by soft tissue radiography. The reproducibility of the component measurements was approximately 1%.

Analysis of the relationship of cardiac frequecy to body muscle has been based on the generally accepted view that in individual subjects the exercise cardiac frequency (fc) is a linear function of oxygen uptake ($\dot{V}o_2$) i.e.

$$fc = A + B\dot{V}o_2.$$

We assume, on the evidence cited in the introduction, that the values of A and B depend to some extent on the quantity of body muscle, both being least for subjects with greatest amount of muscle. The terms may also vary with the state of physical training of the subjects but for our present purpose we have minimized this effect by largely confining the study to subjects who take a moderate amount of exercise. The simplest function which will satisfy the requirement is then an inverse power of the muscle mass and this has led us to test an expression of the form:

$$fc = a + bM^{-\theta}(1 + c\dot{V}o_2) \qquad (1)$$

where a, b, c and θ are constants and M is a measure of body muscle. The parameters a, b and c were estimated by least squares using fixed values of the exponent θ. This was done for a grid of values when it was found that the precise values for θ were not critical: accordingly the optimal values were expressed to the nearest half integer.

Results

The first stage of the analysis showed that the exercise cardiac frequency standardized for consumption of oxygen was negatively correlated with the indices of body muscle and that in Equation 1 the optimal values for θ were unity for lean body mass and total body potassium, 1·5 for thigh muscle width and 0·5 for thigh muscle volume. When muscle was described in terms of lean body mass and thigh muscle width the corresponding values for the coefficient terms a and c were found to be numerically almost equal and the values using potassium and thigh muscle volume were not significantly different. The relationship of exercise cardiac frequency to the indices of body muscle and of lean body mass to thigh muscle width did not differ significantly between men and women or between group A subjects and group B subjects. Accordingly the data were pooled for derivation of optimal values for the coefficient terms a and c. The resulting relationships of cardiac frequency to body muscle are illustrated for lean body mass and for total body potassium in Figs 1 and 2

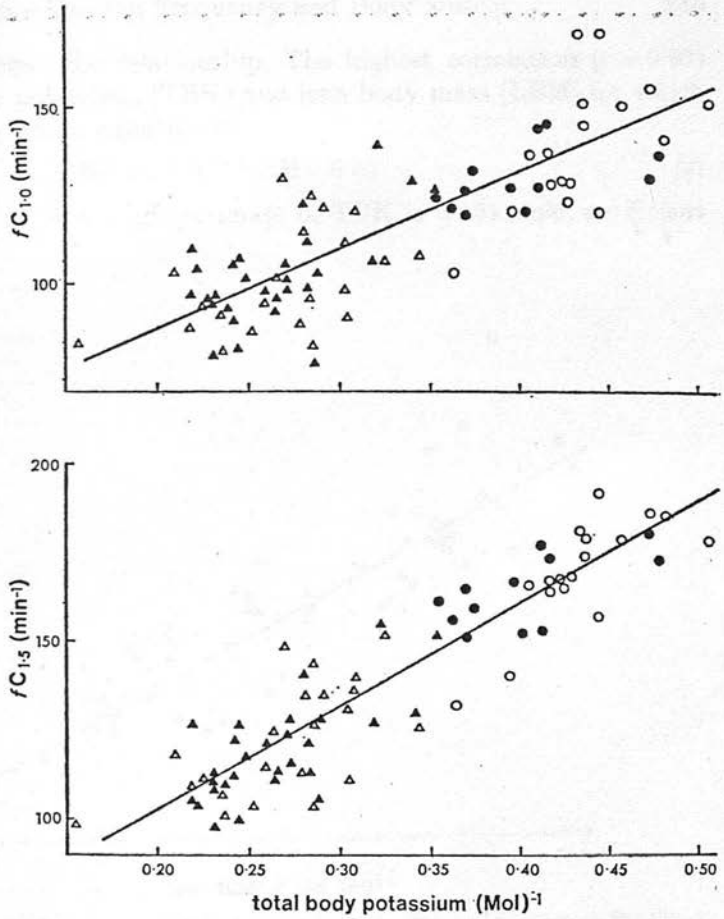

FIG. 2. Relationship to total body potassium of cardiac frequency during submaximal exercise at the oxygen uptakes of 1·0 and 1·5 l. min^{-1} (f $c_{1\cdot0}$ and f $c_{1\cdot5}$ respectively). The symbols are as for Fig. 1. The regression line is constructed using equation 2 (see Table II).

This turns out to be the case for the relationship between thigh muscle width and lean body mass (TMW and LBM respectively) which are accurately described by the equation:

$$0{\cdot}82\ \text{LBM} = \text{TMW}^{1\cdot5}\ \text{(coefficient of variation 7·9\%)} \qquad (3)$$

This relationship is illustrated in Fig. 3.

For the relationships between muscle indices which include as one of the pair either the total body potassium or the thigh muscle volume, the line of best fit differs slightly from that which might be expected from Table II. The observed relationships, like those for cardiac frequency on body muscle, are similar within each group as between men and women and between group A subjects and group B subjects. There is a high degree of inter-correlation between all the indices of muscle ($r \geqslant 0{\cdot}89$), to the extent that in the description of cardiac frequency the inclusion of all four indices of muscle does not materially

reduce the scatter about the relationship. The highest correlation ($r = 0{\cdot}97$) is between total body potassium (TBK) and lean body mass (LBM) for which the corresponding regression equation is:

$$\text{TBK} = 0{\cdot}072\ \text{LBM} - 0{\cdot}57. \qquad (4)$$

The standard deviation of a single estimate of TBK is 0·204 mole, coefficient of variation 6·1%.

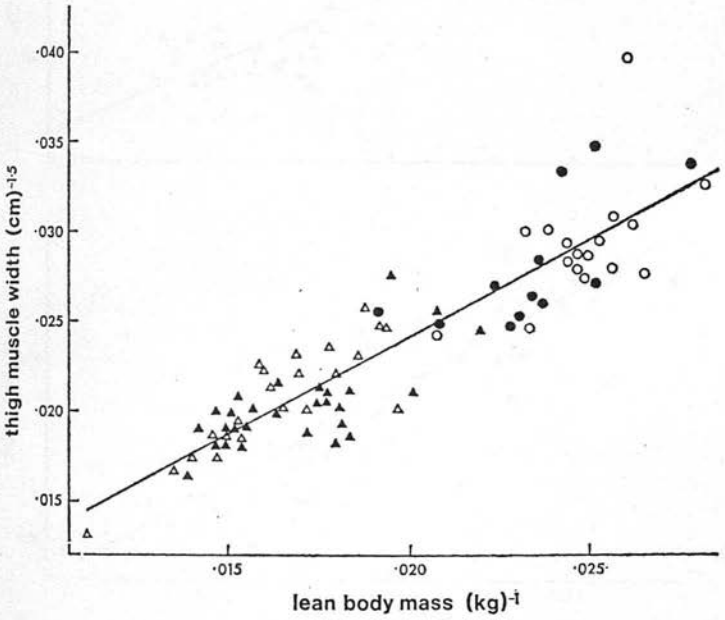

FIG. 3. Relationship of thigh muscle width to lean body mass. The symbols are as for Fig. 1. The regression line is constructed using Equation 3 (see text).

Discussion

The present indices of body muscle were obtained with careful attention to the details of the measurements, so that the reproducibility and accuracy were within acceptable limits (see Methods). The measurements of cardiac frequency and consumption of oxygen was similarly made by reliable methods but in series A the data were only obtained at two rates of submaximal exercise. Thus the finding that nearly identical results were obtained for series B, in which measurements were made in a different laboratory and at a series of work levels, is additional confirmation of the findings. The relationships of cardiac frequency to body muscle are based on data which have a range of at least 2-fold (Table I); the data are distributed symmetrically along the regression lines (Figs 1–3), and the data for men and women overlap. Thus on technical grounds the finding that the data for men and women may be described by common regression lines seems to be unexceptional. In the case of the relationships between thigh muscle width and lean body mass the finding is confirmed by data from Trinidad, where amongst males of Indian and African descent

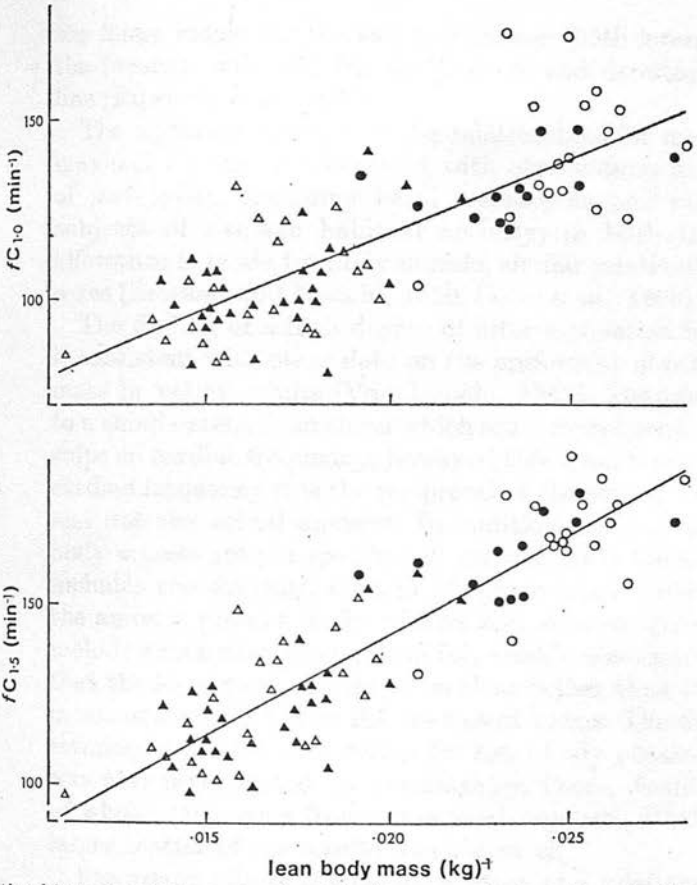

Fig. 1. Relationship to lean body mass of cardiac frequency during submaximal exercise at the oxygen uptakes of 1·0 and 1·5 l. min^{-1} ($f c_{1\cdot0}$ and $f c_{1\cdot5}$ respectively). The circles and triangles refer respectively to women and men; group A subjects are open symbols and group B subjects closed symbols. The regression line is constructed using equation 2 (see Table II).

and the coefficient terms of the regression equation (Eqn. 2) are illustrated in Table 2. The result carries the implication that the several muscle indices should be linked through the respective values for the overall coefficient term M.

TABLE II. *Equations relating exercise cardiac frequency* (f c min^{-1}) *to indices of body muscle*

$f_c = 34\cdot1 + 1300\ M^{-1}\ (1 + 0\cdot0365\ \dot{V}O_2)$ (Eqn. 2)

$\dot{V}O_2$ is consumption of oxygen in m-mole min^{-1}, M is an index of body muscle as follows:

Measurement	Value of M	Coefficient of variation* (%)
Lean body mass (LBM, kg)	0·82 LBM	11·8
Thigh muscle width (TMW, cm)	$TMW^{1\cdot5}$	12·2
Total body potassium (TBK, mole)	14·1 TBK	11·1
Thigh muscle volume (TMV, l.)	$15\cdot5\ TMV^{0\cdot5}$	11·8

*i.e. standard deviation about the regression line expressed as a percentage of the mean value for cardiac frequency (see Table I).

the mean values for the two indices were both intermediate between those for the present male and female subjects and described by the same regression line [Edwards *et al.*, 1972].

The apparent identity of the relationships for men and women during submaximal exercise is consistent with observations at other standardized levels of metabolism including basal metabolism and maximal oxygen uptake in subjects of average habitual activity; in both these circumstances, when allowance is made for body muscle, similar relationships are obtained for both sexes [Astrand and Rodahl, 1970, Cotes *et al.*, 1969].

The finding of a high degree of inter-correlation between the muscle indices is consistent with other data on the uniformity of composition of the lean body mass in young adults [Von Dobeln, 1963]. The observed relationships differ to a small extent from those which are a consequence of the respective relationships on cardiac frequency; however, this is not unexpected, since for estimating cardiac frequency it is the reciprocal of the amount of muscle which is relevant and not the actual amount. In addition the several indices used to describe body muscle are not specific but include other tissues as well. Lean body mass includes the substantial mass of the skeleton, total body potassium includes the amount present in the viscera and nervous system and muscle itself may include connective tissue, deep fat, vessels, tendons, etc. Thus it is remarkable that the agreement should be so close rather than that there should be minor inconsistencies between the coefficient terms. The findings provide a basis for estimation of expected values for total body potassium in young adults. This was also investigated by Burkinshaw, Cotes, Jones and Knibbs [1971], part of whose data came from these same subjects. However, in the later study a larger scatter of the results was observed.

For young adults the demonstration of a relationship between submaximal exercise cardiac frequency and body muscle is evidence both that there is an association between the quantities of cardiac and skeletal muscle and that the cardiac stroke output during exercise is determined to a material extent by the muscularity of the subject. However, the cardiac stroke output is also influenced by other factors, including the extent to which the subject takes strenuous exercise. Athletic training reduces the exercise cardiac frequency without a comparable change in cardiac output, so the cardiac stroke output is increased. The gain in stroke output may be accompanied by an increase in size of the heart, but is mainly due to more complete emptying of the heart during systole [Holmgren, Mossfeldt, Sjostrand and Strom, 1960]. Active subjects have in general a lower cardiac frequency indicating a higher stroke output than those who are sedentary. If active subjects were also more muscular this might explain the association between cardiac frequency and body muscle which we have observed. Two lines of evidence militate against this view. First, the recruitment procedure produced a majority of subjects who were moderately active as rated on a 4-point scale [Cotes *et al.*, 1969], but none who were in athletic training at the time of study. Three of the men had in the past competed in national athletic events, but although their inclusion extended the ranges of the data, their exclusion did not lead to a materially different

result. The activity score for the men was slightly higher than that for the women but the difference was not material. Second, the present results have been compared with those for other groups of subjects of contrasting levels of habitual activity. For amateur racing cyclists compared with the present subjects the thigh muscle width relative to the lean body mass was increased, but only by 4·6%. The exercise cardiac frequency of the cyclists after standardizing for uptake of oxygen was lower than that for the present subjects by an amount which was the same at all levels of body muscle [Cotes, Dabbs, Daley, Hall, Johnson, Reed and Saunders, 1971]. A similar result was obtained for New Guinea highlanders who also took much exercise [Cotes, Adam, Anderson, Kay, Patrick and Saunders, 1972]. In the case of the people studied in the Caribbean the slope of the relationship of cardiac frequency on body muscle was again similar to that for Europeans irrespective of the level of activity of the subjects. However, compared with the present data the cardiac frequencies at all levels of body muscle were displaced upwards for sedentary subjects and downwards for hill farmers amongst whom the level of habitual activity was increased [Miller, Cotes, Hall, Salvosa and Ashworth, 1972]. These observations are summarized in Fig. 4; they support the view that the present result is unlikely to be due to variations between subjects in the levels of habitual activity. Instead the cardiac frequencies of subjects having unusually high or low levels of habitual activity may be described in terms of the additive effects of their endowment with body muscle and the extent to which their habitual activity deviates from that of subjects who are moderately active. Similar considerations may apply to the early stages of cardiovascular disease where the starting point for any deviation from normal cardiac frequency is likely

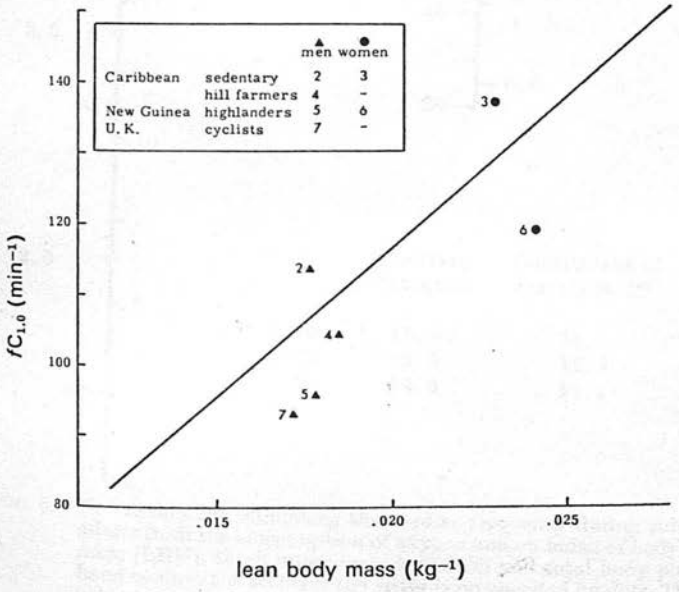

Fig. 4. Relationship of exercise cardiac frequency ($f\,c_{1.0}$) to lean body mass for the present subjects (from Fig. 1) and mean data for groups of subjects having higher or lower levels of habitual activity; for details see text.

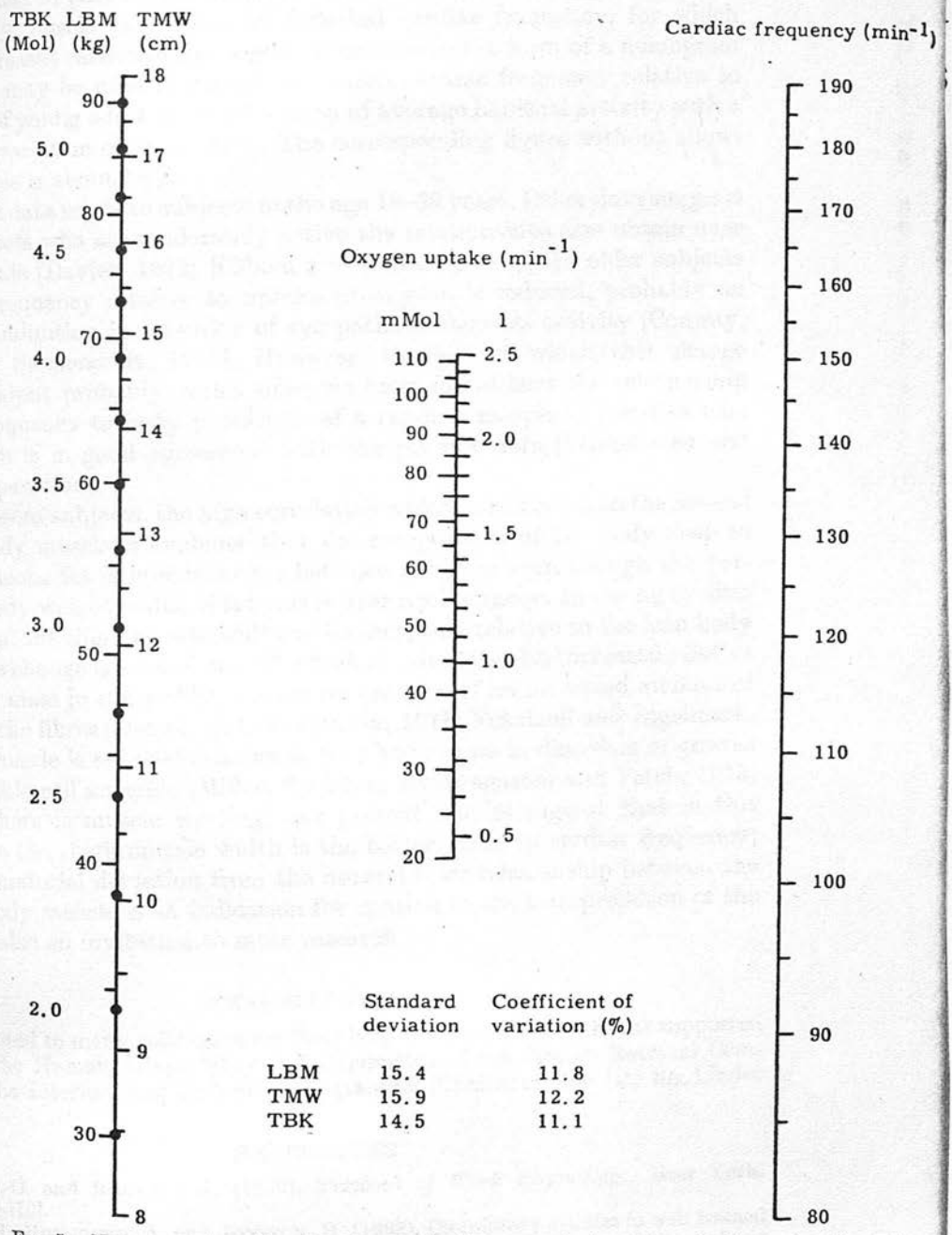

FIG. 5. Nomogram for estimating the cardiac frequency during submaximal exercise of young adults from the consumption of oxygen and an index of body muscle including lean body mass (LBM), thigh muscle width (TMW) and total body potassium (TBK). In the left hand column the scale for the latter is represented by dots. The nomogram is constructed using equation 2 (see Table II).

to be determined in part by the amount of muscle. Thus there may be a place for using body muscle as a guide to expected cardiac frequency, for which purpose the present relationships may be expressed in the form of a nomogram (Fig. 5). This may be used to predict the mean cardiac frequency relative to body muscle of young adult men and women of average habitual activity with a coefficient of variation of about 12%. The corresponding figure without allowance for muscle is about 18%.

The present data relate to subjects in the age 18–38 years. Other data suggests that for subjects who are moderately active the relationships also obtain over the next decade [Davies, 1972; Kilbom and Astrand, 1971]. In older subjects the cardiac frequency relative to uptake of oxygen is reduced, probably on account of diminution in intensity of sympathetic nervous activity [Conway, Wheeler and Sannerstedt, 1971]. However, the age at which this change becomes apparent probably varies since we have found that the relationship of cardiac frequency to body potassium of a random sample of Swedish men aged 54 years is in good agreement with the present data [Wilhelmsen and Cotes, in preparation].

For the present subjects, the high correlation which exists between the several indices of body muscle is evidence that the composition of the body deep to the subcutaneous fat is homogeneous between subjects even though the percentage of body-weight which is fat varies over a wide range. In racing cyclists we have found the thigh muscle width to be increased relative to the lean body mass, but the change is a small one. The bulk of muscle is also increased relative to lean body mass in some older women on account of an increased amount of fat between the fibres [Durnin and Womersley, 1971; Frantzell and Ingelmark, 1951]. The muscle is reduced relative to lean body mass in disorders of growth including sickle cell anaemia [Miller, Serjeant, Sivapragasam and Petch, 1973] and when there is muscle wasting; our present results suggest that in this circumstance the thigh muscle width is the better guide to cardiac frequency; however, a material deviation from the normal inter-relationship between the indices of body muscle is an indication for caution in the interpretation of the result; it is also an invitation to more research.

Acknowledgments

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Effect of treatment on physiological responses to exercise in East African industrial workers with iron deficiency anaemia

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Effect of treatment on physiological responses to exercise in East African industrial workers with iron deficiency anaemia

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Davies, C. T. M., and van Haaren, J. P. M. (1973). *British Journal of Industrial Medicine*, **30**, 335-340. **Effect of treatment on physiological responses to exercise in East African industrial workers with iron deficiency anaemia.** The physiological responses to exercise on an upright bicycle ergometer have been investigated in groups of anaemic (mean Hb = 7·8 g/100 ml) and control (mean Hb = 13·7 g/100 ml) East African industrial workers before and after treatment with oral iron (Fe 200 mg) tablets and on a third group of anaemic subjects (mean = 7·4 g/100 ml) who received no treatment.

Before treatment anaemic subjects exercised with an increased cardiac frequency at a fixed oxygen intake ($\dot{V}O_2$) of 1·5 l/min and a marked reduction in predicted maximum aerobic power output when compared to normal controls. The effect of therapy was to reverse these changes so that no significant differences existed between the two treatment groups but both were significantly different from their anaemic controls. The pulmonary minute ventilation at a $\dot{V}O_2$ of 1·5 l/min and the $\dot{V}O_2$ at a work load of 8 820 Nm/min (900 kpm/min) were similar in all three groups of subjects and remained constant throughout the experimental investigation. The findings carry important implications for those engaged in industrial medicine in developing countries and show clearly that the debilitating effects of iron deficiency anaemia can be rapidly eliminated by simple and inexpensive therapy in factory workers without seriously disturbing their normal work routine.

Anaemia is accompanied by changes in the haemodynamic responses to exercise in man (Sproule, Mitchell, and Miller, 1960; Duke and Abelmann, 1969; Davies, Chukweumeka, and van Haaren, 1973) and in a reduction in maximum aerobic power output (Davies, Chukweumeka, and van Haaren, 1973). The effect of treatment of anaemia on these physiological responses to work is less well documented. Further, compared to the studies at rest, which uniformly show that the disturbances to the cardiorespiratory system due to the anaemia can be rapidly reversed by therapy (Brannon, Merrill, Warren, and Stead, 1945; Backman, 1961; Roy, Bhatia, Mathur, and Virmani, 1963; Duke and Abelmann, 1969) the evidence from the exercise studies available is conflicting. For instance, Andersen and Barkve (1970) show in five patients that the cardiac response and oxygen cost of exercise are reduced following treatment, but both Beutler, Larsh, and Tanzi (1960) and Cotes *et al.*

(1972) show no consistent changes in either exercise tolerance or the cardiorespiratory adaptations to work after therapy. Indeed, Beutler and his co-workers go further and suggest that the symptoms of iron deficiency may arise from mechanisms other than a generalized defect in the O_2 transporting system.

In the present study we have investigated the physiological responses to exercise before and after iron therapy in 22 male adult African industrial workers. A preliminary report of this work has been given (Davies and van Haaren, 1973).

Subjects

The subjects were young adult Africans aged 18 to 35 years living and working in Dar es Salaam, Tanzania. They were identified during a large industrial survey of iron deficiency anaemia (Figure) and subsequently their history was taken and a medical examination performed. Only those subjects who were free from clinical signs of disease, other than the anaemia, were included in the investigation. The design of the experiment was explained to each subject and his consent to take part was obtained.

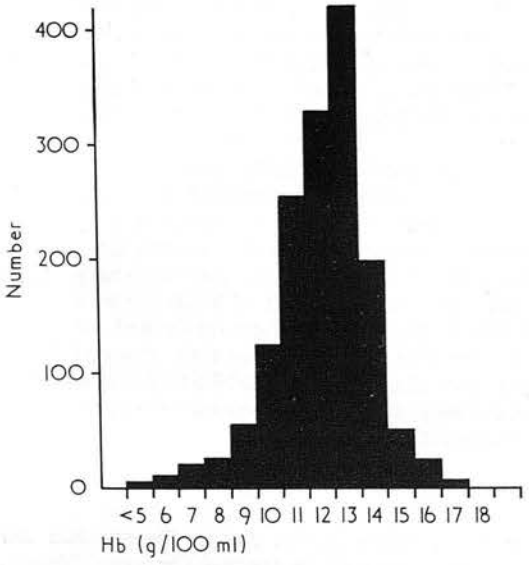

FIGURE. Histogram showing distribution of circulating haemoglobin concentration (g/100 ml) in a survey of industrial workers in Dar es Salaam from whom the subjects of the present investigation were selected. It will be noted that 15% of the industrial population of Dar es Salaam have Hb levels below 10 g/100 ml and 50% have values below 12 g/100 ml, the recommended lower limit for normal healthy subjects by the World Health Organization (1959).

The study was originally made up of 30 subjects who were divided into two groups, normal controls with Hb > 11·5 g/100 ml and anaemic test subjects with Hb < 8·5 g/100 ml. They reported to the laboratory on two separate occasions. On the first visit their responses to exercise were measured: then the test subjects and the normal controls were given daily treatment over a period of approximately three months with tablets containing ferrous sulphate, 200 mg per day. The treatment was supervised each day by the medical assistants at the subjects' factories and a record was kept of attendance.

Of the original 30 subjects taking part in the investigation six normal controls and two anaemic test subjects failed to complete the course of treatment. In addition, five anaemic subjects failed to report for treatment at all, although they were persuaded to attend the laboratory again after three months for their response to exercises to be measured again. Thus, three rather than the original two groupings have been used in this analysis: group I—anaemic test subjects (n = 8), group II—normal controls (n = 9); and group III—anaemic controls (n = 5).

Methods

Details of the methods used to measure the subjects during exercise have been previously given (Davies *et al.*, 1973) and only brief outlines of involved procedures will be given here.

Exercise was performed on a Monark (upright) ergometer. Each subject was allowed to practise and perform the work test on one occasion before definitive measurements of exercise performance were made in an attempt to overcome the problem of habituation to bicycling, as described by Davies, Tuxworth, and Young (1970).

The subjects were required to exercise at five consecutive work loads, the work load being raised by 1 470 Nm/min (150 kpm/min) every two minutes. During the final minute of each work load ventilation minute volume (by Parkinson-Cowan CD4 dry gas meter) and cardiac frequency (from a standard Philips cardiograph) were recorded, and expired air was collected from a mixing chamber for subsequent analysis.

In 8 of the 22 subjects, expired air was analysed for O_2 and CO_2 content using a paramagnetic O_2 analyser (Servomax Ltd.) and a Katharometer (Cambridge Instruments Ltd.) respectively, and oxygen intake ($\dot{V}O_2$) was calculated in the usual way. However, in the remaining 14 subjects CO_2 content was not measured and $\dot{V}O_2$ was estimated from minute ventilation volume and O_2 content using the Weir (1949) formula and a standard calorific value for oxygen of 21·0 kJ/litre (5 kcal/litre). This method of calculating $\dot{V}O_2$ is likely to introduce an additional 2% error (Passmore and Draper, 1963) into energy expenditure measurements and should be borne in mind when interpreting our results. Individual regression lines were fitted to the $\dot{V}O_2$, minute ventilation ($\dot{V}_E$), and cardiac frequency (f_H) data which enabled $\dot{V}_E$ and f_H to be expressed in terms of a given $\dot{V}O_2$ of 1·5 l/min ($\dot{V}_E$ 1·5 and f_H 1·5) (Cotes *et al.*, 1969).

Similarly, regression of $\dot{V}O_2$ on work output ($\dot{W}$) and $\dot{V}O_2$ on f_H were calculated for each subject and from these equations $\dot{V}O_2$ was expressed at a given $\dot{W}$ of 8 820 Nm/min (900 kpm/min) ($\dot{V}O_{2\ 900}$) and maximum

aerobic power output ($\dot{V}O_{2max}$) was predicted by extrapolation of the $\dot{V}O_2/f_H$ curve to an assumed $f_{H\,max}$ of 195 (Davies, 1968; Ojikutu, Fox, Davies, and Davies, 1972).

In addition, weight and the skinfold thickness at four sites (biceps, triceps, subscapular, and suprailiac) were measured, from which lean body mass (LBM) was calculated after the method of Durnin and Rahaman (1967). Blood haemoglobin concentration was measured by the cyanmethaemoglobin method. Duplicate measurements were required to agree within ± 0·5 g/100 ml.

Results

The physical characteristics of the test and control subjects are given in Table 1. The test subjects are slightly lighter in weight, shorter in stature, and have less body fat than the normal controls, but none of the differences shown in Table 1 except for circulating blood haemoglobin reaches conventional levels of significance.

The responses to exercise before treatment are summarized in Table 2. They show that for a fixed work load of 8 820 Nm/min (900 kpm/min) there is no significant difference in $\dot{V}O_2$ between the three groups. This is also true for minute ventilation ($\dot{V}_{E1\cdot5}$), but there is a marked tendency towards higher mean values in the two anaemic groups (Davies *et al.*, 1973). The cardiac frequency at $\dot{V}O_2$ of 1·5 l/min ($f_{H1\cdot5}$) is significantly lower ($P < 0\cdot05$) and the predicted maximum aerobic power ($\dot{V}O_{2max}$) is significantly higher ($P < 0\cdot05$) in the normal controls compared with the two anaemic groups.

Effects of treatment

Following treatment with oral iron tablets (Fe 200 mg), there is a marked and significant increase (+ 5·6 g/100 ml $P < 0\cdot001$); in blood haemoglobin in the test subjects, the haemoglobin of the two control groups remaining unchanged. The mean changes in the physiological responses to exercise (Table 3) show that the major effect of treatment was a marked and significant decrease (−23 beats/min ($P < 0\cdot001$)) of $f_{H\,1\cdot5}$ in test subjects compared to the normal (−1 beat/min) and anaemic controls (−7 beats/min). There was

TABLE 1

PHYSICAL CHARACTERISTICS OF SUBJECTS: AGE, WEIGHT, HEIGHT, THE SUM OF 4 SKINFOLD THICKNESSES (BICEPS, TRICEPS, SUBSCAPULAR, AND SUPRAILIAC), LEAN BODY MASS, CALCULATED FROM SKINFOLD THICKNESSES AFTER THE METHOD OF DURNIN AND RAHAMAN (1967), AND CIRCULATING BLOOD HAEMOGLOBIN

Group	*Age* (*yr*)	*Wt* (*kg*)	*Ht* (*cm*)	Σsf (*mm*)	*LBM* (*kg*)	*Hb* (*g/100 ml*)
Test subjects I (n = 8)	22·0 ±6·3	50·3 ±5·1	162·1 ±4·5	20·4 ±2·8	45·8 ±4·3	7·8 ±1·2
Normal controls II (n = 9)	26·0 ±3·5	56·3 ±8·8	165·4 ±5·7	24·3 ±6·5	50·2 ±7·9	13·7*** II, III ±1·1
Anaemia controls III (n = 5)	24·4 ±8·1	54·5 ±7·6	161·7 ±4·8	25·6 ±1·8	48·1 ±6·6	7·4 ±1·0

Significant differences: *** $P < 0\cdot001$

TABLE 2

RESPONSES TO SUBMAXIMAL EXERCISE EXPRESSED IN TERMS OF OXYGEN INTAKE AT A WORK LOAD OF 8 820 nm/min (900 kpm/min) ($\dot{V}O_{2\,900}$) AT MINUTE VENTILATION AT A $\dot{V}O_2$ OF 1·5 l/min ($\dot{V}_{E\,1\cdot5}$), CARDIAC FREQUENCY AT A $\dot{V}O_2$ OF 1·5 l/min ($f_{H1\cdot5}$) AND PREDICTED MAXIMUM AEROBIC POWER ($\dot{V}O_{2max}$)

Group	$\dot{V}O_{2\,900}$ (l/min)	$\dot{V}E_{\,1\cdot5}$ (l/min)	$f_{H1\cdot5}$ (beats/min)	$\dot{V}O_{2max}$ (l/min)
Test subjects I (n = 8)	2·06 ±0·16	54·5 ±14·7	159 ±14·7	2·05 ±0·32
Normal controls II (n = 9)	2·06 ±0·16	45·2 ±7·4	136** I,III ±12	2·54* I,II ±0·41
Anaemia controls III (n = 5)	2·05 ±0·08	52·8 ±4·4	165 ±14	1·95 ±0·19

Significant differences: *$P < 0\cdot05$; **$P < 0\cdot01$

TABLE 3

EFFECTS OF TREATMENT ON PHYSICAL CHARACTERISTICS AND HAEMOGLOBIN CONCENTRATION

Group	*Wt* (*kg*)	$\sum sf$ (*mm*)	*LBM* (*kg*)	*Hb* (*g*/100 *ml*)
Test subjects I (n = 8)	+2·24	+2·54	+2·67	+5·6*** II,III
Normal controls II (n = 9)	−0·14	+1·31	−0·41	+0·4
Anaemic controls III (n = 5)	+0·50	−6·06*I, II	+1·97	0·5

Significant differences—see Tables 1 and 2

corresponding increase in predicted $\dot{V}O_{2max}$ of + 530 ml/min (26%; P < 0·01) in test subjects compared to + 70 ml/min and + 150 ml/min in the other two groups respectively.

There was a non-significant increase in $\dot{V}O_2$ 900 of + 150 ml/min common to all three groups; $\dot{V}_{E1\cdot5}$ was reduced (−6·2 l/min) in the test subjects, remained constant in the normal controls, and increased slightly (+ 1·8 l/min) in the anaemic subjects who did not receive treatment. None of the latter changes reached acceptable levels of significance (Table 4).

Body dimensions remained the same in groups I and II. In the anaemic subjects who did not receive treatment (group III) there was a significant mean decrease of 6·1 mm in skinfold thickness (P <0·05).

Discussion

The results confirm our previous findings (Davies *et al.*, 1973) that anaemia (Hb < 8·5 g/100 ml) gives rise to increased cardiac frequency for a given $\dot{V}O_2$ without a concomitant change in either the minute ventilation or the O_2 cost of work during submaximal exercise (Table 2). At higher levels of work anaemia results in a marked reduction in predicted maximum aerobic power output. The data further show that these changes can be reversed by approximately three months of daily oral iron therapy. These results confirm the findings of Backman (1961) and Duke and Abelmann (1969) at rest and the relevant observations of Andersen and Barkve (1970) in exercise, but they are at variance with the work of Beutler *et al.* (1960) and Cotes *et al.* (1972). However, both these latter studies are difficult to interpret from the treatment point of view. Beutler and his co-workers (1960) studied only four 'anaemic' patients, two of whom had Hb concentrations in excess of 12 g/100 ml before treatment was given, and Cotes and his colleagues were unable to demonstrate any differences between their test subjects and controls before treatment was started. In our experiment we took four major precautions: (1) we tried to ensure that our subjects were familiar with and habituated to the work (Davies *et al.*, 1970) so that the confounding influence of short-term changes in the responses to exercise due to increased familiarity with the test procedures was minimized; (2) we attempted to study our subjects over the complete range of their work capacity up to and occasionally including maximum levels of work in order to minimize the large intra-subject variation in the

TABLE 4

EFFECTS OF TREATMENT ON RESPONSES TO EXERCISE

Group	$\dot{V}O_{2\ 100}$ (l/min)	$\dot{V}_{E1\cdot5}$ (l/min)	$f_{H\ 1\cdot5}$ (beats/min)	$\dot{V}O_{2max}$ (l/min)
Test subjects I (n = 8)	+0·15	−6·20	−23*** II	+0·53*** II
Normal controls II (n = 9)	+0·16	−0·04	− 1	+0·07
Anaemic controls III (n = 5)	+0·15	+1·80	− 7	+0·15

Significant differences—see Tables 1 and 2

prediction of $\dot{V}O_{2max}$ which occurs when using the $\dot{V}O_2/f_H$ relationship (Davies, 1968); (3) we supervised the daily taking of the oral iron tablets (this is important in Africa where climate greatly affects the condition of the tablets and where the general population is unused to taking tablets); and (4) we encouraged all our test and control subjects to continue their normal daily (industrial) tasks during the period of the treatment. However, it is unlikely that (1) contributes significantly to the differences we have found between the present study and that of Cotes *et al.*, since in both investigations control subjects were used and any differences due to habituation or learning in the exercise test would presumably be reflected equally in the anaemic, normal and control subjects. Equally, (3) is unlikely to be a relevant factor to the two investigations because in European countries the climate is less harsh and the general population more readily accept the idea of taking tablets or pills for preventive as well as curative purposes. The difficulties of persuading Africans to take tablets and accept treatment when they have no visible or outward signs of illness is underlined by the present study and suggests that this may be an important problem which must be overcome if effective treatment is to be given to African populations on a community basis. Although we were aware of the problem and took the precautions outlined, nevertheless 13 of the original 30 subjects (43%) failed either to complete or to begin our course of treatment.

This leaves precautions (1) and (2), and we feel that these two factors undoubtedly had some influence on our results. First, as we have shown previously (Davies *et al.*, 1973), it is only at higher levels of work that the differences between anaemic and non-anaemic subjects become readily apparent. This may have enabled us to demonstrate clear differences in the test and control groups initially and show the effects of treatment more clearly. Secondly, measurements on two severely anaemic patients undergoing treatment in hospital (Davies and van Haaren, unpublished observations) did not demonstrate any significant improvement in their responses to exercise despite increases of approximately 50% and 100% in circulating haemoglobin concentration. It would appear that in these patients the potential for exercise had been restored, but their actual work performance had remained unaltered. This puts the question of habitual levels of activity into sharp focus in the treatment of anaemia. The fact that our subjects were leading 'normal' lives, performing everyday tasks, and not confined to hospital wards may account in part for the large changes in physiological function we have been able to demonstrate.

Clearly, the responses to different severities of test exercise in anaemic subjects and the effects of habitual activity combined with therapy are areas for future studies. The results of this investigation do, however, clearly demonstrate that the debilitating effects of anaemia can be reversed by appropriate treatment in subjects who continue to perform their normal daily work.

This finding has an important bearing on a developing country, such as Tanzania, where iron deficiency is endemic (Figure) and where social and economic development depend on human labour. Provided the problems of supervision can be overcome to ensure that the iron tablets are taken regularly over a period of time and workers are encouraged to remain or become more active, then a serious loss of work output and reduced productivity (Davies, 1973) which is associated with anaemia, may be avoided at minimal cost to the occupational health service and the community.

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PERCEIVED EXERTION DURING RHYTHMIC EXERCISE INVOLVING DIFFERENT MUSCLE MASSES

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The perceived exertion (RPE) and maximal and submaximal responses to one-arm, two-arm, one-leg and two-leg work performed on a suitably modified, stationary, bicycle ergometer have been studied on six healthy male subjects. The results show that the RPE is closely associated with work output (W), minute ventilation rate ($\dot{V}_E$), oxygen intake ($\dot{V}_{O_2}$), and cardiac frequency (f_H), but for a given individual and for the group as a whole, the degree and form of the association is dependent on the type of exercise performed and the limb (muscle plus bone) volume involved. The differences in RPE/W, $\dot{V}_E$, $\dot{V}_{O_2}$ and f_H relationships, however, could be removed by standardising for the maximal aerobic power output ($\dot{V}_{O_2max}$) of the subjects attained in the different work forms. This suggests that relative 'aerobic' stress is an important determinant of RPE and together with cues based on previous experience it may form the basis for the way in which a given subject accurately perceives his own exertion during rhythmic exercise with one or two limbs on a bicycle ergometer.

Borg and his co-workers (BORG, 1962; BORG and LINDERHOLM, 1967; SKINNER *et al.*, 1969; BORG, 1970; BAR-OR *et al.*, 1972) have developed and validated a numerical scale for rating the perceived exertion of exercise. The scale is based on bicycle ergometer exercise and is constructed to give a linear increase of rated perceived exertion with heart rate. It has been suggested that such a scale might have useful industrial and clinical applications both as a measure of the stress involved in a given task and as a predictive device for estimating physical working capacity (BORG, 1970; EDWARDS *et al.*, 1972; GAMBERALE, 1972). However, it is not clear which physiological changes are primarily responsible for the perception of exertion, although a number of possibilities have been suggested (HENRIKSSON *et al.*, 1972; EKBLOM and GOLDBERG, 1971; EDWARDS *et al.*, 1972).

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Furthermore, whilst the scale has been developed from stationary bicycle ergometer exercise involving both legs, many everyday tasks involve only a relatively small muscle mass (*e.g.* one or both arms) where the physiological responses to exercise at the same relative work loads ($\%\dot{V}_{O_2}$ max) may be quite different (DAVIES and SARGEANT, 1973a and b). Thus in order to assess the potential application of the RPE scale to tasks involving different muscle groups, we have examined the relationship between rated perceived exertion and physiological responses to single and double limb exercises in the laboratory under standardised conditions.

MATERIALS AND METHODS

Six healthy male subjects, aged 24–39 years, took part in the investigation. Their physical details are given in Table 1. All of the subjects had taken part in physiological investigations previously and all were habituated to the forms of exercise involved in this study (see DAVIES *et al.*, 1970).

Table 1. Age (year), weight (kg), height (cm), limb (muscle plus bone) volumes (liter) and maximal aerobic power outputs ($\dot{V}_{O_2}$ max liter/min) for work with the right arm (RA); two arms (2A); right leg (RL) and two legs (2L).

Subject	Age (year)	Weight (kg)	Height (cm)	Limb volumes (liter)				$\dot{V}_{O_2}$ max (liter/min)			
				RA	2A	RL	2L	RA	2A	RL	2L
1	28	73.3	179.3	2.30	4.39	7.20	14.40	0.99	1.53	2.28	3.21
2	39	63.0	177.3	1.84	3.24	6.33	12.82	0.82	1.20	2.01	3.10
3	31	98.0	183.0	3.24	6.47	9.20	17.60	1.60	1.97	2.78	3.69
4	24	77.3	176.5	2.10	4.33	8.19	16.37	1.50	1.78	2.59	4.00
5	27	84.4	189.0	—	—	7.77	15.52	—	1.99	2.62	3.22
6	35	78.5	171.8	—	—	8.56	17.13	—	1.78	2.86	3.62

Four types of exercise were studied, they were (in order of increasing muscle mass involvement), one-arm, two-arm, one-leg and two-leg exercises. The exercises all involved pedalling with the legs or cranking with the arms at a frequency of 50 revolutions per minute on either a standard or modified Monark bicycle ergometer. In one-arm and one-leg work, both the right and left limbs were measured on separate occasions and the data from each limb included in the results. Careful attention was paid to standardisation procedures, particularly in one- and two-arm work where movement was restricted as far as possible to the arms alone by strapping the subject onto a backboard. Further, details of these procedures are given in previous reports from this laboratory (DAVIES and SARGEANT, 1973a and b).

The subjects exercised for six minutes at each of 5 work loads in the 4 different exercises studied. The range of work loads were: 0–250 kpm/min

(1-arm); 0–600kpm/min (2-arm); 0–850kpm/min (1-leg) and 0–1500kpm/min (2-legs). Measurements of oxygen intake and heart rate were taken over the last two minutes of each exercise intensity. Oxygen intake ($\dot{V}_{O_2}$) was measured by a continuous system (DAVIES, 1968) using a Parkinson Cowan CD4 gas meter, Servomex Paramagnetic and Beckman LBI gas analysers for ventilation volume, oxygen and carbon dioxide concentrations respectively. The aerobic energy expenditure during work ($\dot{V}_{O_2}$ net) was calculated from a baseline of zero load (see WHIPP and WASSERMAN, 1969). Cardiac frequency (f_H) was measured using lightweight chest electrodes and a differential ECG amplifier connected in series to a linear instantaneous rate meter (Devices Sales Ltd).

Rated perceived exertion was assessed using the revised scale proposed by BORG (1970) giving scale values from 6 to 20, each odd number on the scale chart being accompanied by a brief verbal description, as given below:

6	
7	very very light
8	
9	very light
10	
11	fairly light
12	
13	somewhat hard
14	
15	hard
16	
17	very hard
18	
19	very very hard
20	

Subjects were shown the chart at the end of each six minute exercise session and asked to indicate a number on the scale corresponding to the degree of exertion perceived.

Limb volume (LV) measurements were made after the method of JONES and PEARSON (1969) adapted in the case of the arms as previously described by DAVIES and SARGEANT (1973a).

Maximum aerobic power ($\dot{V}_{O_2}$ max) of the subjects performing the different forms of exercise were measured directly on separate occasions using a standard Douglas bag technique (DAVIES, 1968). All expired gas volumes were expressed at STPD and ventilatory volumes at BTPS. The criteria used for ascertaining the $\dot{V}_{O_2}$ max during the different forms of exercise have been described in detail elsewhere (DAVIES and SARGEANT, 1973a and b).

RESULTS

There is a significant linear relationship ($P<0.05$) between rated perceived exertion (RPE) and work load (W), minute ventilation ($\dot{V}_E$), cardiac frequency (f_H) and oxygen intake ($\dot{V}_{O_2}$) for the different exercises studied (Table 2), but

Table 2. Relationships of y (dependent variable)to RPE (x) in the four forms of exercise studied characterised by: a, constant term; b, regression coefficient; p, significance; SEY, standard error of estimate of y; r, correlation coefficient; and n, number.

Exercise	y	a	b	p	SE_y	r	n
2 legs		−848	94.9	0.001	167	0.904	17
1 leg	$\dot{V}_E$ net	−813	83.8	0.001	145	0.890	57
2 arms	ml/kg/min	−452	50.1	0.01	139	0.804	14
1 arm		−441	44.8	0.001	87	0.887	25
2 legs		−650	103.0	0.001	189	0.896	17
1 leg	W	−401	60.5	0.001	96	0.905	56
2 arms	kpm/min	−251	39.4	0.01	113	0.807	14
1 arm		− 74	17.7	0.001	45	0.821	23
2 legs		18.3	8.22	0.001	20.0	0.837	17
1 leg	f_H	10.0	7.73	0.001	14.7	0.872	57
2 arms	beats/min	5.8	7.22	0.02	25.7	0.651	14
1 arm		33.7	5.55	0.001	16.0	0.787	25
2 legs		−0.91	0.19	0.001	0.40	0.873	17
1 leg	$\dot{V}_{O_2}$	−0.89	0.16	0.001	0.28	0.883	57
2 arms	liter/min	−0.44	0.11	0.01	0.27	0.853	14
1 arm		−0.15	0.07	0.001	0.22	0.755	25
2 legs		−22.7	5.26	0.001	10.5	0.881	17
1 leg	%	−39.8	6.73	0.001	8.78	0.933	57
2 arms	$\dot{V}_{O_2}$ max	−19.0	5.25	0.001	5.06	0.904	14
1 arm		−21.3	5.79	0.001	5.99	0.964	25

the form of the regression relation is dependent on the type of work performed and the degree of muscle mass involvement (Fig. 1). The progressive increase in active limb (muscle plus bone) volume involvement during one-arm, two-arm, one-leg and two-leg work (Table 1) is associated with a corresponding rise in the predicted maximal levels of W (from 280 kpm/min in 1-arm to 1,410 kpm/min in two-leg work), $\dot{V}_E$ (0.45 to 1.05 liter/$kg_{(body\ weight)}$/min), f_H (145 to 183 beats/min) and $\dot{V}_{O_2}$ (1.25 to 2.89 liter/min) at an RPE of 20. Most of these differences, however, can be removed by standardising (Fig. 2) for the maximal aerobic power output ($\dot{V}_{O_2}$ max). If the net $\dot{V}_{O_2}$ (*i.e.* $\dot{V}_{O_2}$ during exercise minus $\dot{V}_{O_2}$ pedalling at zero load—see methods) is expressed in relative terms as a % of the $\dot{V}_{O_2}$ max the only significant difference ($P<0.025$) remaining is between the slope of the regression lines for two-leg and one-leg work. Although even here the regression

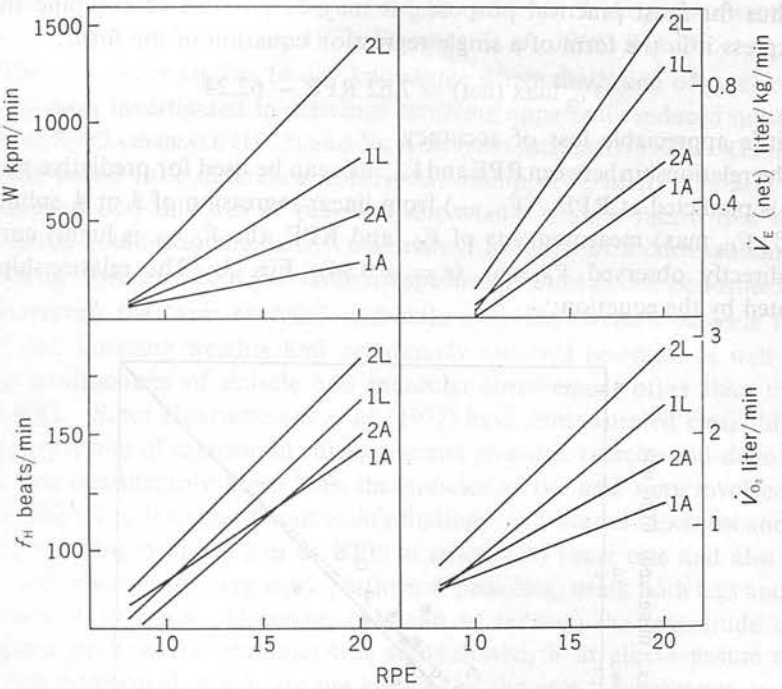

Fig. 1. Relationship of work load (W), minute ventilation ($\dot{V}_E$), cardiac frequency (f_H), and oxygen intake ($\dot{V}_{O_2}$) to rated perceived exertion (RPE). Regression lines given for two-leg (2L), one-leg (1L), two-arm (2A), and one-arm (1A) exercise.

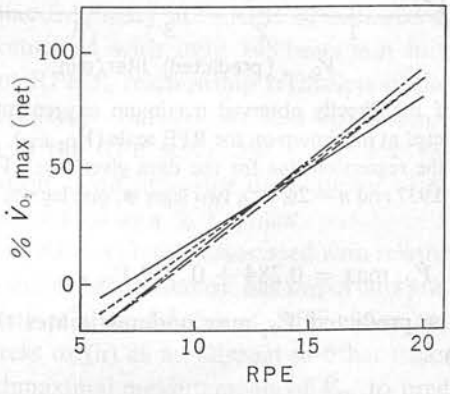

Fig. 2. Relationship of relative work load (% $\dot{V}_{O_2}$ max) to rated perceived exertion for two-leg (——), one-leg (-·-), two-arm (----) and one-arm (--) exercise.

lines intersect at ~RPE 15 and the difference in predicted % $\dot{V}_{O_2}$ max even at the extremes of the scale, *i.e.* RPE 6 and 20 is only about 10%.

Thus for most practical purposes, it may be possible to combine the data and express it in the form of a single regression equation of the form:

$$\% \dot{V}_{O_2} \text{ max (net)} = 7.62 \text{ RPE} - 62.24$$

with little appreciable loss of accuracy.

The relationship between RPE and $\dot{V}_{O_2}$ max can be used for predictive purposes. If $\dot{V}_{O_2}$ is predicted at RPE_{20} ($\dot{V}_{O_2\,R20}$) from linear regression of 3 or 4 submaximal ($<80\%$ $\dot{V}_{O_2}$ max) measurements of $\dot{V}_{O_2}$ and RPE, the $\dot{V}_{O_2\,R20}$ is highly correlated with directly observed $\dot{V}_{O_2}$ max ($r = +0.94$), Fig. 3. The relationship is represented by the equation:

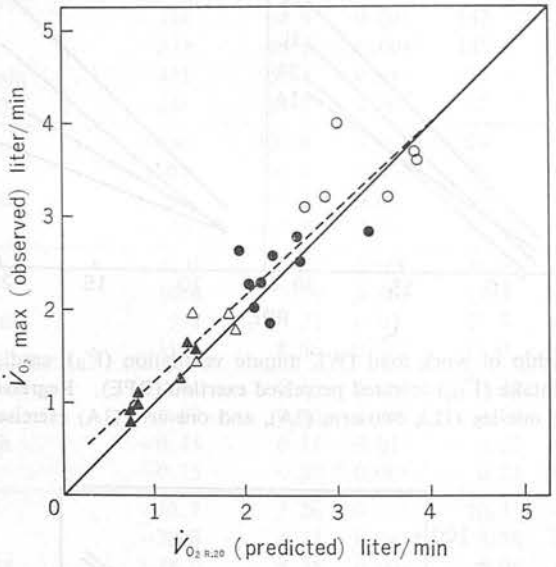

Fig. 3. Relationship of the directly observed maximum oxygen intake ($\dot{V}_{O_2}$ max) to the oxygen intake predicted at maximum on the RPE scale ($\dot{V}_{O_2\,R20}$). The line of identity is shown along with the regression line for the data given by: $\dot{V}_{O_2}\text{max} = 0.284 + 0.929\ \dot{V}_{O_2\,R20}$ where $r = 0.937$ and $n = 26$. ○, two legs; ●, one leg; △, two arms; ▲, one arm.

$$\dot{V}_{O_2} \text{ max} = 0.284 + 0.929\ \dot{V}_{O_2\,R20}$$

Using this equation the predicted $\dot{V}_{O_2}$ max underestimates the observed $\dot{V}_{O_2}$ max by $-6\% \pm 13\%$ (SD)

DISCUSSION

Few data are available on the rating of perceived exertion in standardised exercise when the active muscle mass is reduced, and our findings of linear relationships between RPE and $\dot{V}_E$, W, $\dot{V}_{O_2}$, f_H and $\% \dot{V}_{O_2}$ max in exercise involving

one arm, two arms, and one leg extend the previous findings for two leg work (BORG, 1970; EDWARDS *et al.*, 1972; SKINNER *et al.*, 1969; BAR-OR *et al.*, 1972).

The only other studies to our knowledge where the rating of perceived exertion has been investigated in activities involving apparently reduced muscle mass are those by GAMBERALE (1972) and EKBLOM and GOLDBERG (1971). Gamberable, however, found little difference in the relationship of f_H to RPE in arm exercise and claimed that this was of practical importance since it meant that the RPE scale rating could be immediately understood in terms of a corresponding heart rate, being close to 1/10th the value irrespective of the exercise performed.

However, the 'arm exercise' undertaken by Gamberale's subjects involved lifting and lowering weights and presumably involved eccentric as well as concentric contractions of muscle and muscular involvement other than the arms (*e.g.* back). Since HENDRIKSSON *et al.* (1972) have demonstrated clear differences in the perception of exertion in concentric and eccentric exercise and since it seems likely that considerably more than the muscles of the arm were involved, it becomes difficult to interpret Gamberale's findings. Conversely EKBLOM and GOLDBERG (1971) found differences of RPE in relation to heart rate and also relative work load when comparing work performed pedalling, using both legs and cranking, using both arms. However, they fail to indicate the magnitude of these differences or how the cranking was standardised, if at all, to ensure that the work was performed mainly by the muscles of the arm. In contrast our results based on exercises which were carefully standardised and restricted as far as possible to specific limb activity involving concentric rhythmic muscle activity (See DAVIES and SARGEANT, 1973a and b), indicate clear and progressive differences between the relationship of RPE to W, $\dot{V}_E$, $\dot{V}_{O_2}$ and f_H. Indeed in the latter case predicted cardiac frequency at ~RPE 20 indicates a level of 183 beats/min in two-leg work compared with only 145 beats/min in one-arm work. Hence the use of a constant RPE/f_H relationship regardless of the type of activity undertaken cannot be supported. Further our data would suggest in contrast to Borg's original conception of the RPE scale, that the critical variable for relating RPE in the applied situation as an indicator or predictor of exercise stress, is relative work load, *i.e.* $\dot{V}_{O_2}$ expressed as a $\%\ \dot{V}_{O_2}$ max.

The evidence that RPE is closely associated with relative work load irrespective of the muscle mass involved in exercise has important practical implications since it emphasises the potential usefulness of RPE either as (i) a tool in itself for assessing relative stress or (ii) as an adjunct to other measurements, for example, when linked with submaximal measurements of $\dot{V}_{O_2}$ to predict $\dot{V}_{O_2}$ max: this latter technique may prove particularly valuable in specific activities such as arm work where conventional predictions based on the $\dot{V}_{O_2}/f_H$ relationship of two-legged exercise (*e.g.* Åstrand and Rhyming) are no longer valid (Davies and Sargeant, unpublished observations). The $\dot{V}_{O_2}$ max predictions for our subjects based on submaximal (<80%) RPE/$\dot{V}_{O_2}$ measurements give a mean error of $-6\% \pm 13\%$

(SD) which is similar to that found in two-leg bicycle exercise using conventional $\dot{V}_{O_2}/f_H$ extrapolation techniques (DAVIES, 1968).

However, it must be pointed out that these findings related to the prediction of $\dot{V}_{O_2}$ max from submaximal $\dot{V}_{O_2}$ and RPE need to be treated with some caution since although they are based on 26 separate direct determination of $\dot{V}_{O_2}$ max (of right and left legs and arms, and both legs, and both arms) these were obtained on a fairly homogenous (in terms of activity, habituation and interest) group of only six subjects. The potent influence of interest and motivation on perception generally is well known (see VERNON, 1962 for general review) and presumably applies equally to the perception of exertion. Before our results can be applied on a wider basis to the industrial and medical fields, there is a need for further research to extend the findings to larger groups, of individuals selected at random from the community.

Finally, the wide variations in the maximal levels attained in W, $\dot{V}_E$, f_H, and $\dot{V}_{O_2}$ depending on the amount of muscle mass involved suggests that no one of these physiological variables is an overriding cue for the perception of exertion.

It has been suggested (GAMBERALE, 1972) that the degree of anaerobic metabolism as reflected in the plasma levels of lactic acid (LA) may play an important role in the perception of exertion; this could hardly account, however, for the accurate perception of exertion at low work levels. Furthermore, data collected on two of our subjects indicate plasma LA levels approximately 15 mgs% higher for given relative work loads above 50% $\dot{V}_{O_2}$ max in two-arm compared with two-leg work (DAVIES and SARGEANT, 1973a), which should on this account lead to higher RPE ratings in arm work, but our present findings fail to indicate any such differences. Similarly data collected on the levels of plasma catecholamines (DAVIES *et al.*, 1973) indicate lower values of circulatory catecholamines in one-leg compared with two-leg work at any given relative work load. This would seem, therefore, to discount the implication of these substances, as indicators of circulatory stress or associated mechanisms, from any dominant role in the perception of exertion.

We are therefore, like EDWARDS *et al.* (1972), HENRIKSSON *et al.* (1972) and others drawn to the conclusion that there is an integration of a number of factors involved which is compared subconsciously against previous experience. If this is so one might expect differences between groups dependent on the extent and depth of previous experience and again points to the need for more data on different and larger groups.

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The Physiological Responses to Running Downhill

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Abstract. The metabolic cost of one healthy male subject aged 31 years, running on a range of gradients from + 5 % to − 45 % at speeds varying from 6.0 km · h^{-1} to 18.0 km · h^{-1}, on a motor-driven treadmill, has been investigated.

The results showed that the "apparent" efficiency of running increased with gradient from 0 to − 15 % and then remained fairly constant at a value similar to that found for downhill walking (see Davies and Barnes [6]) of − 1.2 until a gradient of − 35 % was reached. Beyond this gradient, there was a further rise in efficiency to reach − 1.41 at − 45 %. The speed of running was controlled by stride length from 0 to − 20 %, but at higher gradients there was an increase in step frequency which was speed dependent. The relationships of $\dot{V}_E/\dot{V}_{O_2}$ and $f_H/\dot{V}_{O_2}$ were similar to those previously decribed for downhill walking except that at gradients > − 15 % there was a parallel displacement of the $f_H/\dot{V}_{O_2}$ to the right. The relationship of $\dot{V}_{O_2}$ to speed of running uphill and downhill was essentially linear and thus for both forms of exercise, for a given gradient, the aerobic cost of running per unit distance covered was constant and independent of speed.

Key words: Negative Work — $\dot{V}_{O_2}$ — Eccentric Contraction — Efficiency.

Introduction

The kinetics of level and uphill running, in which energy is expended to raise and lower the centre of gravity and accelerate and decelerate the limbs have been extensively investigated [2, 4, 9, 10, 12, 15—17]. In contrast little attention has been paid to activities, namely downhill work, where the energy necessary for progression is not provided by the body but by gravitational forces. In these circumstances, provided the runner can stride freely (i.e. little work is performed to overcome frictional resistance or the gravitational pull is not too great to necessitate the forcible deceleration of the limbs) the energy expenditure should be at a minimum. Thus the "apparent" mechanical efficiency (defined as the ratio of positive or negative work performed to the aerobic energy expended) should be close to if not exceeding other forms of downhill activity such as walking [6, 13] and laddermill climbing [11]. It is there-

fore surprising to find [13] that the efficiency of downhill running is 60% lower than for walking down a given decline and only just over twice as efficient as walking or running uphill.

In the present investigation we have studied intensively on one healthy male subject the physiological cost of downhill running in relation to speed, gradient and mechanical work output.

Material and Methods

The data presented here was collected on a healthy male subject (B.S. age: 31 years, weight: 61.1 kg, height: 178 cm). The subject was carefully habituated to both running and walking downhill [5, 8] and allowed to become familiar with the procedures involved and was then studied over a period of 6 months at 92 different work levels.

The subject usually reported to the laboratory in the afternoon following a light lunch. After a brief initial warm-up run on the treadmill he was fitted with the necessary electrodes and respiratory apparatus and exercised for 6 min at the chosen speed and gradient; measurements being taken over the last 2 min of exercise.

A similar but less comprehensive series of experiments were performed on a second subject DB; although the data were less consistent they supported the conclusions drawn from the results of BS. Since the study on DB was incomplete (due to pressure of examinations) we have not included his data in the present analysis.

Cardiac frequency was continuously monitored from chest electrodes connected to an ECG differential amplifier and linear instantaneous ratemeter (Devices Sales Ltd.). Oxygen intake was measured by an open circuit technique previously described [6] where inspiratory volume was measured with a CD 4 dry gas meter (Parkinson-Cowan Ltd.) and O_2 and CO_2 concentrations with Servomex and Beckman meters respectively. Both meters were regularly calibrated with gases of known concentration as determined by the Haldane method.

Results

In downhill work beyond a certain speed it was found that it was more efficient to run than walk (Fig. 1). In our subject at gradients of -25% and -30% this speed was $\sim 7.5\ \mathrm{km \cdot hr^{-1}}$ (cf. Boje [2] in positive work). The relationship of energy expenditure (E: calculated from $\dot{V}_{O_2}$) to positive or negative mechanical work output (W: calculated from the overall distance that the body weight is raised or lowered respectively) over the range of gradients and at a given speed is shown in Fig. 2. The lines radiating from the origin represent the iso-efficiency values. For a given speed energy expenditure decreases with gradient from 0% to -15% and then rises again in such a way that the mechanical efficiency values of running downhill between -15% and -35% approach a level of -1.2 (Fig. 2) which is closely similar to that found for downhill walking at lower speeds [6]. However, beyond -35% the calculated value of energy expenditure remains fairly constant despite

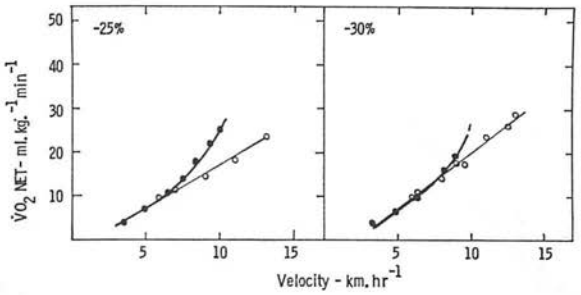

Fig. 1. The relationship of net oxygen intake ($\dot{V}_{O_2}$ net) to velocity at – 25 % and – 30 % gradients. Walking (●), running (○)

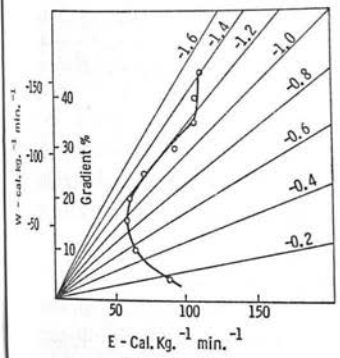

Fig. 2

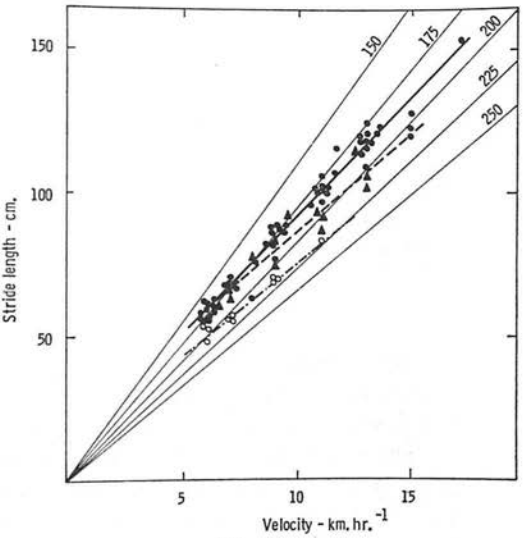

Fig. 3

Fig. 2. The relationship of work performed (W) to energy expenditure (E) for gradients – 5 % to – 45 % and a given speed of 9 km·h^{-1}. Iso-efficiency lines are shown

Fig. 3. The relationship of stride length (S_L) to velocity of running. ●——● +5 % to – 20 %, ▲- - - -▲ – 25 % and –30 %, ○·····○ – 35 % to – 45 %

an increase in the amount of negative work performed so that the efficiency of the work shows a further rise to reach values of — 1.29 and — 1.41 at — 40 % and — 45 % respectively. For a given energy expenditure from 0 % to — 20 %. step frequency remained fairly constant; in-

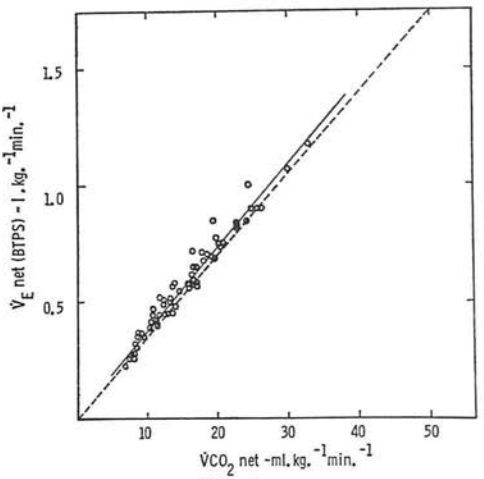

Fig. 4

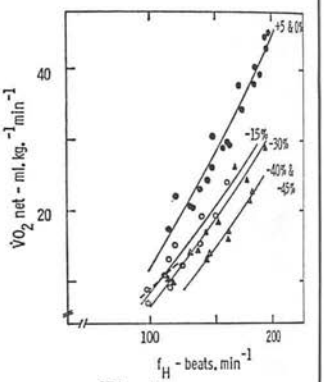

Fig. 5

Fig. 4. Net minute ventilation ($\dot{V}_E$ BTPS) in relation to carbon dioxide output ($\dot{V}_{CO_2}$). ○——○ Present data—downhill running; - - - - - downhill walking (from Davies and Barnes [5, 6])

Fig. 5. The relationship of net oxygen intake ($\dot{V}_{O_2}$ net) to cardiac frequency (f_H). Subject BS running (present data): ●——● + 5 % and 0 %, ○——○ −15 %, ▲——▲ − 20 %, △——△ − 40 % and − 45 % gradients. Subject BS walking (from Davies and Barnes [6]): - - - - - − 25 % to − 35 % gradients

creased velocity (as in positive work) was achieved by increased stride length. However, beyond — 20 % there was an increase in step frequency which was dependent on running speed (Fig. 3).

The pulmonary minute ventilation rate at a fixed $\dot{V}_{O_2}$ of 1.5 l · min^{-1} ($\dot{V}_{E\,1.5}$) is independent of gradient from — 5 % to — 35 % but at the higher two loads there is a marked ($\sim$ 10 l · min^{-1}) and a significant increase in $\dot{V}_E$. The $\dot{V}_{E\,1.5}$ is higher in downhill than in uphill running but these differences disappear if $\dot{V}_E$ is related to $\dot{V}_{CO_2}$ (cf. Davies and Barnes [6]). The relationship of $\dot{V}_E$ to $\dot{V}_{CO_2}$ is linear (Fig. 4) over the range studied and can be represented by the equation:

$$\dot{V}_E \text{ net} = 0.036\ \dot{V}_{CO_2} \text{ net} + 0.024\ .$$

The linear relationship between exercise cardiac frequency (f_H) and $\dot{V}_{O_2}$ is similar to that found for walking downhill if the gradient in running does not exceed — 15 % (Fig. 5); beyond this level there is a parallel shift of the $\dot{V}_{O_2}/f_H$ line to the right. At a $\dot{V}_{O_2}$ of 1.5 l · min^{-1} the f_H rises from 128 beats min^{-1} at 0 % to 165 beats min^{-1} at — 45 % and there is a concomitant fall in predicted maximum aerobic power ($\dot{V}_{O_2\,max}$) of $\sim$ 33 % from 2.92 l · min^{-1} at 0 % to 1.96 l · min^{-1} at — 45 %.

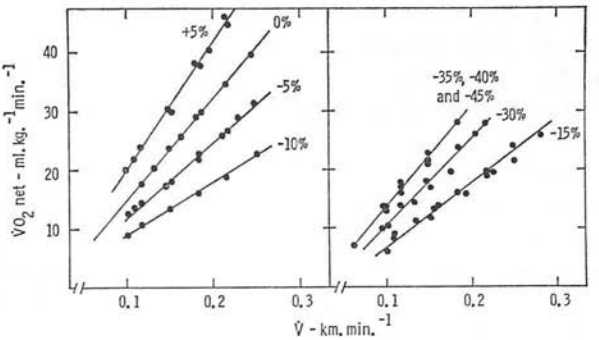

Fig. 6. The relationship of net oxygen intake ($\dot{V}_{O_2}$ net) to velocity (v) of running on inclines from + 5% to − 45%

In Fig. 6 the relationship of the $\dot{V}_{O_2}$ of running (ml · kg^{-1} min^{-1}) to velocity (km · min^{-1}) is given.

At all levels of exercise in both positive and negative work $\dot{V}_{O_2}$ appears to be a linear function of velocity. Thus for a given gradient the O_2 cost of running per km of distance covered is constant and independent of speed. On the level the net aerobic cost of running approximates to 190 ml · kg^{-1} km^{-1} at + 5% this increases to 218 ml · kg^{-1} km^{-1} (cf. Margaria *et al.* [15]). From 0% to — 15% the $\dot{V}_{O_2}$ net per kilogram body weight and per kilometre distance covered gradually decreases to a value of 100 ml but beyond this gradient level $\dot{V}_{O_2}$ net begins to rise and reaches a value of 175 ml at — 45%.

Discussion

This study was primarily undertaken to establish the efficiency and physiological responses to downhill running performed on a motor driven treadmill. The results suggest that provided the speed is > 7.5 km · h^{-1} and the gradient is > — 15% and < — 35% running downhill is performed with the same efficiency as walking at lower speeds, namely — 1.2 (see Fig. 2 and Davies and Barnes [6]). However, at very steep inclines of the treadmill bed the efficiency of running is 20% higher than that for walking or running at lower slopes and is of the order of — 1.41 (Fig. 2).

The physiological responses to running are also similar to those previously given for walking. For given $\dot{V}_{CO_2}$ the $\dot{V}_E$ is identical in these two forms of exercise (Fig. 4); the regression equations relating $\dot{V}_E$/ $\dot{V}_{CO_2}$ in walking (see Davies and Barnes [6]) and running downhill are for all practical purposes interchangeable. This is also true for the $\dot{V}_{O_2}/f_H$

relationship for gradients below — 15% (Fig. 5); however, beyond this level of exercise there is a parallel displacement of the $\dot{V}_{O_2}/f_H$ line downwards, i.e. for a given $\dot{V}_{O_2}$, f_H is higher under these conditions than in walking. Thus the differences previously described for negative and positive work [6] are further exaggerated if downhill running at very steep gradients is considered.

Our data for the energy cost of downhill running are at variance with the findings of Margaria [13] and Margaria *et al.* [15]. In the latter paper they report (p. 369, Fig. 3) a value of mechanical efficiency of — 0.60 for gradients of — 15% and — 20%. This figure is approximately half that found at the same gradients in the present study (Fig. 2). A difference of this magnitude is difficult to interpret and we have no immediate explanation to offer, except that in Margaria's original paper no mention was made of habituation procedures for his subjects. We have shown previously [5] that during repeated daily downhill walking efficiency increases by — 0.5 from day 1 to — 1.2 at day 5. This improvement of efficiency during a period of habituation to negative work, if it can be applied to running, would clearly explain in part the differences in energy expenditure between the two studies. Certainly, in our experience downhill running is an activity requiring skill which can only be achieved through practice. During initial runs on the treadmill, the subjects are apprehensive and tend to perform many waste movements. In particular, they appear to overcompensate for the forward motion of the body by an exaggerated braking action of the leading leg; consequently not only is extra energy dissipated in this way but quite often the pull of gravity becomes insufficient to provide the force necessary for forward motion and the subject must perform an additional amount of work to maintain his position with respect to the treadmill bed. This large discrepancy between Margaria's and the present study supports our view [5, 6] and those of Kamon [11] that a set value of efficiency cannot be used for negative work unless the conditions are clearly defined and stated.

However, some caution is necessary in trying to interpret measurements of $\dot{V}_{O_2}$ in terms of energy cost in this type of exercise and indeed in level and uphill running. During positive work the $\dot{V}_{O_2}$ is only a valid measure of energy requirements provided that the subject is in a steady state and the anaerobic component of work is minimal. It is well-known that at high work outputs the $\dot{V}_{O_2}$ plateaus as the level of $\dot{V}_{O_2\,max}$ is reached and there is an increase in anaerobiosis. Thus one might expect to see a levelling off rather than a disproportionate rise in $\dot{V}_{O_2}$ at very high running speeds, particularly on an incline. Under these conditions $\dot{V}_{O_2}$ will not be representative of the true energy cost of the positive work performed. In negative work the situation is more complex still, since during this type of exercise the muscles (particularly the quadri-

ceps) become stretched during the contracted phase as a result of gravitational and inertial forces and thereby develop more force [18]. Part of this work done on the muscle may be either stored in the series elastic component [31]. Under these conditions the $\dot{V}_{O_2}$ will not reflect the total energy exchange within the muscle. These factors will of course also have a direct bearing on the term efficiency as used in this study.

We have calculated the "apparent" mechanical efficiency of our runner throughout the experiments as the ratio of work performed to energy expended. The values of these two parameters have been derived from O_2 cost and the mechanical work output due to treadmill inclination, body weight and speed. The word "apparent" is important because our method of calculation (i) involves only the aerobic cost of work (as indicated by the $\dot{V}_{O_2}$) and does not include other possible forms of energy utilization within the muscle cited above and (ii) though it excludes rest, it takes no account of the energy cost of simply maintaining the upright posture necessary to perform the run. On a bicycle ergometer the postural cost of cycling can be determined by pedalling at zero load. On the treadmill it is probably best estimated by a backward extrapolation of the $\dot{V}_{O_2}/v$ curve to calculate the $\dot{V}_{O_2}$ at zero velocity. In the present experiments this value is $\sim 3.5\,\mathrm{ml \cdot kg^{-1} min^{-1}}$ for both negative and positive work (Fig. 6). If this figure is subtracted from the energy values shown in Fig. 2 then the "apparent" efficiency will increase. In positive work (running uphill + 5% gradient) the effect will be negligible ($< 2\%$) but in downhill running the increase in apparent mechanical efficiency will be of the order of 15 to 20%.

Finally, it has been suggested by some authors [2, 12, 17] that the relationship of $\dot{V}_{O_2}$ net to velocity during uphill or level running is curvilinear. We, however, like Margaria *et al.* [15], found no reason to suspect a breakdown in the linear relationship of uphill or level running, and our data indicates a similar linearity in downhill running.

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Physiological Responses to Standardised Arm Work

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The physiological responses of 8 healthy male subjects were studied during one- and two-arm cranking exercise performed on a suitably modified bicycle ergometer under carefully standardised conditions utilising a shoulder restraining board and harness designed to restrict trunk movement.

'Apparent' mechanical efficiency (E—defined as the ratio of work performed to aerobic energy expended) was found to be similar at low levels of exercise to that previously found for leg work, namely 0·25; although there was a fall in E at levels in excess of $\sim 40\%$ $\dot{V}_{O_2\,max}$. $\dot{V}_E$ was higher in arm work than in leg work for a given $\dot{V}_{O_2}$ or $\dot{V}_{CO_2}$: $\dot{Q}$, however remained the same for a given $\dot{V}_{O_2}$, although stroke volume was reduced and cardiac frequency increased in arm work when compared with leg work.

The maximum aerobic power of the arms was found to be $\frac{1}{3}$ that of the legs, but for a given limb volume (muscle plus bone—measured after the method of Jones and Pearson (1969)) was $\sim 13\%$ higher in arm than in leg work: the reasons for this difference are discussed.

1. Introduction

The physiological responses to work with the arms have been studied many times (e.g. Collett and Liljestrand 1924, Christensen 1931, Asmussen and Nielsen 1947, Bobbert 1960, Asmussen and Hemmingsen 1958 and Åstrand and Saltin 1961). All these studies appear to show that work with the arms is mechanically less ($\sim 10\%$) efficient and (where measured) produces a maximum aerobic output ($\dot{V}_{O_2\,max}$) which is $\sim 35\%$ less than similar exercise with the legs. These results are surprising when one considers that (i) children, who have very small leg muscle mass (indeed almost identical to the arm muscle mass of adults) pedal the bicycle ergometer with exactly the same 'apparent' mechanical efficiency as adults (Godfrey *et al.* 1971 and Davies 1973a), and (ii) since the volume of active muscle mass has been shown to be closely associated with $\dot{V}_{O_2\,max}$ (Davies *et al.* 1972, Davies *et al.* 1973) one would not expect the arms, which have an estimated muscle plus bone volume $\frac{1}{3}$ of that of the legs, to produce a $\dot{V}_{O_2\,max}$ $\frac{2}{3}$ that of the lower limbs. However, although several authors have stressed the need for standardisation of body position during work with the legs (see Åstrand and Rohdal 1970) particularly with respect to saddle height and handle bar position and body movement, these important aspects of ergometry seem to have been ignored (or at least not emphasised) when arm, as opposed to leg cranking, exercise has been studied.

In the present investigation we have studied the physiological responses to one- and two-arm cranking exercise on 8 healthy male subjects, aged 24–39 years on a suitably modified stationary bicycle ergometer and we have paid special attention to standardisation procedures; particularly to limiting trunk movement during arm work by use of a shoulder support and harness. Estimations of arm (muscle plus bone) volume have been made after the method of Jones and Pearson (1969) and these have been related to measurements of the aerobic power output of the upper limbs.

2. Material and Methods

The physical details of the 8 subjects who took part in the investigation are given in Table 1. All the subjects were familiar with physiological investigations but none had performed arm work in the way described below before the beginning of the present study.

Table 1. Physical characteristics and maximal aerobic power output ($\dot{V}_{O_2}$ max) of the subjects. Age, weight (Wt), height (Ht), arm (AV) and leg (LV—muscle plus bone) volume and $\dot{V}_{O_2}$ max with the right (RA), left (LA) and both arms (RA+LA) and both legs (RL+LL)

Subject	Age yr	Wt kg	Ht cm	Limb volume–l		$\dot{V}_{O_2}$ max net–l/min			
				AV	*LV*	RA	LA+	RA+ L.A.	RL+ L.L.
AJS	28	73·3	179·3	4·39	14·40	0·65	0·62	1·16	2·85
CTMD	39	63·0	177·3	3·24	12·82	0·53	0·43	0·90	2·73
WP	31	98·0	183·0	6 47	17·60	1·19	1·23	1·52	3·30
JS	24	77·3	176·5	4·33	16·37	0·53	0·89	1·05	3·58
MC	26	77·5	184·0	4·80	17·43	0·78	0·74	1·31	3·90
JF	42	72·2	176·0	4·03	—	—	—	1·71	—
BS	33	63·9	177·2	3·60	—	—	—	0·87	—
MB	29	62·2	172·1	4·50	—	—	—	1·17	—
Mean	31·5	73·4	178·2	4·42	15·72	0·74	0·78	1·14	3·27
SD	6·3	11·7	4·0	0·97	2·06	0·27	0·30	0·21	0·49

Arm Cranking

A modified Monark bicycle ergometer was used to perform the arm exercise. The saddle of the bicycle was removed and was replaced with a shoulder board and harness which allowed the trunk and upper body (except the arms) to be restrained and remain stationary during submaximal and maximal exercise. The ' pedals ' of the crank were replaced with modified ' handle grips ' to prevent rotation of the arm and unnecessary shoulder movement during exercise. ' Control ' experiments were performed with shoulder board and harness removed and for comparison some data were collected during leg work using the same ergometer.

Five of the eight subjects were measured during left arm, right arm, combined arm and combined leg work, on separate occasions over the full range of their exercise capacity from zero load up to and including maximal effort. The remaining three subjects only performed combined arm work (see the Table).

Criteria for Maximal Performance

The criteria which have been developed for leg work (see Davies 1968) were not easy to apply. The majority of subjects did not show a clear plateau of $\dot{V}_{O_2}$ with increasing work at maximal effort due to a small change of mechanical efficiency (see Figure 2 and text) and furthermore, cardiac frequency was always well below that found for the legs. In order to overcome this difficulty we took duplicate measurements of maximal work performance on separate occasions (see Davies and Sargeant 1973).

Cardio-Respiratory Measurements

Oxygen intake ($\dot{V}_{O_2}$), minute ventilation ($\dot{V}_E$), cardiac (f_H), respiratory (f_R), and pedal (f_P) frequencies were measured using methods previously

described (Davies 1968). Mechanical efficiency (defined as ratio of work output to aerobic energy expenditure) was calculated using a baseline of zero load (see Hill 1965 and Whipp and Wasserman 1969).

Cardiac output was measured using a CO_2 rebreathing technique (Jones *et al.* 1967) as previously described (Godfrey and Davies 1970, Godfrey *et al.* 1971). In two subjects (CTMD and AJS) venous blood was sampled during exercise from an antecubital vein and later analysed for lactic acid using the enzymatic method of Gerchen (1960).

Anthropometry

Arm (muscle plus bone) volume was determined by physical anthropometry after the method described for the legs by Jones and Pearson (1969). The arm was treated as a series of truncated cones, the circumferences and lengths being measured with the limb in the horizontal extended position at the following sites: (i) minimal wrist, (ii) maximum forearm, (iii) olecranon process at the elbow joint, (iv) minimum upper arm below biceps, (v) mid upper arm, (vi) upper arm above the biceps and (vii) at the angle of the axilla. A correction for fat based on skinfold thickness measured at the dorsal and ventral forearm surfaces and the biceps and triceps (see Jones and Pearson 1969) was made using equations developed by Jones (1970). Validation of our method for measuring arm volumes (AV) in this way was obtained by water displacement. The regression equation relating AV (by water displacement) to AV (by physical anthropometry) is given by:

$$AV \text{ (displacement)} = 0{\cdot}26 + 0{\cdot}9\ AV \text{ (anthropometry)}.$$
$$\text{SD} = 111 \text{ ml.} \quad r = +0{\cdot}98.$$

3. Results

The 'apparent' mechanical efficiency (E) of one- and two-arm work is shown in Figure 1.

At mean energy expenditure levels below 20 cal kg^{-1} min^{-1}, E is identical in the two forms of work and closely similar to the figure of 0·25 previously found for exercise with the legs. At higher work levels, however, there is a decline in E of $\sim 5\%$ in 2-arm work, and $\sim 9\%$ in 1-arm work.

The minute ventilation rate ($\dot{V}_E$) for a given $\dot{V}_{O_2}$ is significantly ($p < 0{\cdot}001$) higher in arm than in leg work. The increased $\dot{V}_E$ is almost entirely due to a rise in respiratory frequency; $\bar{V}_T$ of arm work being lower than in similar exercise with the legs. When $\dot{V}_E$ is related to $\dot{V}_{CO_2}$ the difference between arm and leg work is reduced, but not eliminated. The association of $\dot{V}_E$ with $\dot{V}_{CO_2}$ is essentially curvilinear for one- and two-arm work and may be represented by the equation:

$$\dot{V}_E \text{ net} = -28{\cdot}58 + 38{\cdot}5\ (\dot{V}_{CO_2}) - 0{\cdot}11\ (\dot{V}_{CO_2})^2.$$

The cardiac output ($\dot{Q}$) for a given $\dot{V}_{O_2}$ is identical in one- and two-arm work and closely similar to that found for work with the legs. However, in arm work cardiac frequency (f_H) is significantly higher ($p < 0{\cdot}001$) than found at comparable levels of energy expenditure in leg work giving rise to a reduction in cardiac stroke volume (S.V.—Figure 2).

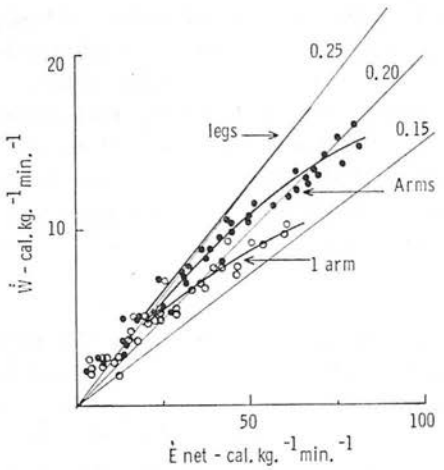

Figure 1. Relationship between work performed and aerobic energy expended. Isopleths for mechanical efficiency are given. Symbols: one Arm (○); two Arm (●).

The blood lactic acid concentration (LA) was also markedly elevated for given $\dot{V}_{O_2}$ in arm compared with leg work but in relative terms (expressed as a $\%\dot{V}_{O_2\,max}$), the LA was significantly lower in the former compared to latter type of exercise. At maximal work in two subject (see Materials and Methods), LA reached approximately 80 mg/100 ml of blood in arm cranking.

Maximum Aerobic Power

The maximum aerobic power ($\dot{V}_{O_2\,max}$) of the arms is approximately $\frac{1}{3}$ of that of the legs in cranking exercise. The relationship of $V_{O_2\,max}$ to arm (muscle plus bone) volume is shown in Figure 3.

The equation relating the two variables is given by:

$$\dot{V}_{O_2\,max}\,(l/min) = 0{\cdot}173 + 0{\cdot}254\,AV\,(l)$$
$$r = +0{\cdot}89 \quad SD = 135\ ml.$$

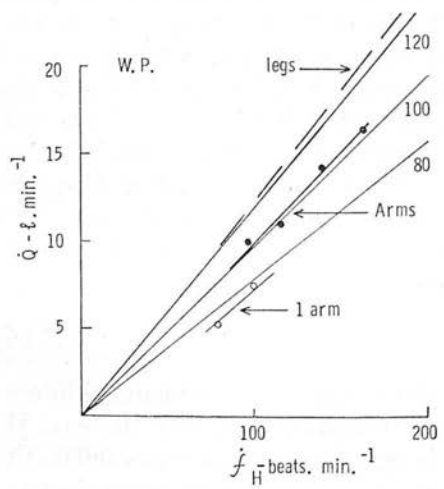

Figure 2. Cardiac output in relation to cardiac frequency. Isopleths for stroke volume are given. Symbols: as for Figure 1.

The regression relationship is displaced to the left of the line found previously for the adult leg work (Figure 3). Thus for given muscle plus bone volume in adults the $\dot{V}_{O_2\,max}$ of the arms is approximately 19% higher than that found for the legs. It is also interesting to note, that for the 5 subjects who performed exercise with one and two arms, the sum of the $\dot{V}_{O_2\,max}$ for two arms measured separately is significantly greater than for the two together.

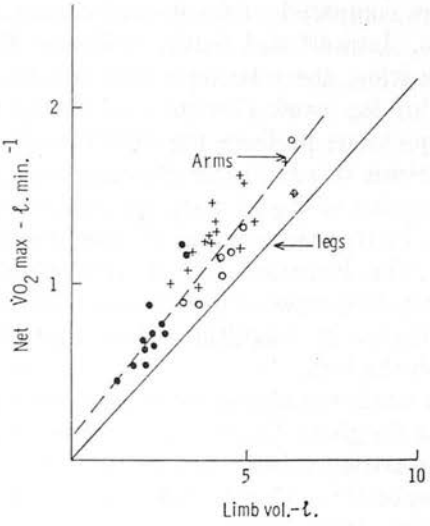

Figure 3. Maximum aerobic power output ($\dot{V}_{O_2\,max}$) in relation to arm volume (AV). 1 Arm (○); 2 arms (●); and leg work in young boys (+ — see text and Davies *et al.* 1970).

4. Discussion

The data of the present study were collected on normal healthy male subjects who were familiar with laboratory experimental procedures. They were required to practise the arm cranking exercise before definitive measurements were taken in order to overcome the possible confounding influences of learning and habituation to the task (see Davies *et al.* 1970). Their results, therefore, may be considered normal for the conditions under which they were taken. The findings suggest that when body position is standardised, the mechanical efficiency of the arms is only marginally lower than the legs in cranking exercise and in arm work (as previously shown for the legs—Davies *et al.* 1973), the maximum aerobic output of one or two limbs is closely associated with the effective muscle plus bone mass used to perform the task.

In arm exercise at aerobic energy expenditure levels < 20 cal kg^{-1} min^{-1} the calculated mechanical efficiency was similar to that found previously for leg work (Figure 1). However, at higher work outputs, there was a disproportionate increase in $\dot{V}_{O_2}$ for a given $\dot{W}$ and thus a decline in mechanical efficiency. The reasons for this fall in efficiency at the higher work loads is not altogether clear. The most likely explanation is a small rise, despite our standardisation procedures, in the postural component of the work at maximum effort. It will be noted that in Figure 3 the line relating $\dot{V}_{O_2\,max}$ to arm (muscle plus bone) volume has a positive intercept of 173 ml min^{-1}. If the assumptions regarding the calculation of mechanical efficiency and $\dot{V}_{O_2\,max}$ from a baseline of

zero load are correct (see Whipp and Wasserman 1969) this phenomenon can only be interpreted as a small increase in the postural component of work necessary to sustain the arm cranking movements at maximal effort. If a value of 173 ml min^{-1} is subtracted from the $\dot{V}_{O_2}$ cost of arm exercise at the highest work loads, the difference in mechanical efficiency of leg and arm cranking using a stationary cycle ergometer disappears.

The findings of an increased f_H for given $\dot{V}_{O_2}$ and a rise of $\dot{V}_E$ for a given $\dot{V}_{O_2}$ or $\dot{V}_{CO_2}$ during arm compared with leg work confirms the work of several authors (Bobbert 1960, Åstrand and Saltin 1961 and Bevegard *et al.* 1963). In the present investigation, the relationship of $\dot{V}_E$ to $\dot{V}_{CO_2}$ in arm work is curvilinear as found for leg work (Davies and Sargeant 1973). However, this differs from our previous findings for uphill and downhill walking and running on a motor driven treadmill (Davies and Barnes 1972, Davies *et al.* 1973) where the relationship of $\dot{V}_E$ to $\dot{V}_{CO_2}$ was essentially linear. This may in part be a reflection of the wider range of exercise intensities used in the present experiments. The increased $\dot{V}_E$ of arm work was achieved by a marked rise in respiratory frequency at work levels > 50% $\dot{V}_{O_2\,max}$, $\bar{V}_T$ remained fairly constant and smaller in magnitude than that found for comparable exercise intensities with the legs.

The rise in f_H in arm work was also accompanied by a fall in cardiac stroke volume; cardiac output for given $\dot{V}_{O_2}$ was independent of the type of exercise performed. The fall in stroke volume in arm compared with leg work in our subjects was of the order of 10% (15 ml). This compares favourably with figures given by Bevegård *et al.* (1963) and Stenberg *et al.* (1967), and is probably related to the inadequacy of the muscle pumps and a fall in venous return which may accompany work with small muscle groups (Bevegård and Shepherd 1967). The values of stroke volume of adults working with the arms reported in the present study are very similar to those found for children working with the legs (Godfrey *et al.* 1971).

Maximal Performance

The data given in the table clearly show that the $\dot{V}_{O_2\,max}$ of one arm is proportionately greater than the $\dot{V}_{O_2\,max}$ of two arms which in turn is $\frac{1}{3}$ that of the legs. However, standardisation for muscle plus bone volume and the small postural component (173 ml/min) of work markedly reduces the difference in $\dot{V}_{O_2\,max}$ between one and two arms, but in relative terms ($ml.l_{(LV)}^{-1}\ min^{-1}$) the arms have a ~13% higher $\dot{V}_{O_2\,max}$ than the legs. This phenomen of an increased $\dot{V}_{O_2\,max}$ per unit muscle (plus bone) volume in the arm work is in our view of physiological importance and may be related to $\dot{Q}$ and the available blood flow to working muscles in the two situations.

During exercise with large muscle groups, such as the legs, it has often been suggested (Bevegård and Shepherd 1967) that the major limit to aerobic exercise is provided by $\dot{Q}$, work beyond a certain level merely results in an increase in anaerobic metabolism and varying degrees of arterial anoxaemia (Rowell *et al.* 1964). In restrained arm work, though there may be an increase in anaerobic metabolism, the same limiting factors are unlikely to apply. From the data given in Figure 2 and the table, it is unlikely that $\dot{Q}$ exceeded 17 l/min^{-1} during our maximum arm experiments, thus together with the subjective responses from our subjects at maximal effort it is reasonable to

suppose that limits to performance were peripheral rather than central in origin. Under these conditions the present experiments suggest that the arm muscle plus bone volume is capable of utilising ~13% more oxygen than the legs in comparable forms of work. This result throws an interesting light on the recent controversies (see Gollnick *et al.* 1972) concerning the limiting factors to exercise.

The relatively high $\dot{V}_{O_2\,max}$ per unit of muscle plus bone volume in adult arm work may also have some bearing on the nonlinearity of the curve relating $\dot{V}_{O_2\,max}$ to age, in children and young adults (Davies *et al.* 1971). Young children (7–9 years) who had leg volumes comparable with the arm volumes of adults (Figure 3) in the present study were found to have a 15% higher relative $\dot{V}_{O_2\,max}$ than their adolescent counterparts.

Finally, we should emphasise that our results take no account of the differences in the proportion of muscle to bone volume in the upper and lower limbs. Clearly the proportion of muscle per bone volume may have an important bearing on our results, but (due to a lack of a suitable method for measuring bone volume) in the present investigation we have made no attempt to separate the two and have expressed our data always in terms of a total muscle plus bone volume. Furthermore, our data have been collected on normal healthy, but in the main, sedentary subjects and care should be taken not to apply the results to groups of subjects in which the level of physical activity may be different or unknown. In previous studies of the relationship between indices of body composition and aerobic power output (Davies *et al.* 1973 and Davies 1973b) it was shown that whilst in homogenous populations normally active but not in training, estimates of limb volume may account for 80% of the variance of $\dot{V}_{O_2\,max}$, the relationship between the two variables may not be regarded as causal. Results from subjects in training and patients with iron deficiency anaemia suggest that limb (muscle plus bone) volume may be varied independently and thus the latter cannot be used for predictive purposes. It is reasonable to assume that similar conclusions apply to estimates of arm (muscle plus bone) volume. In some circumstances (e.g. rehabilitation work involving injury to the lower limbs) where arm work is used as a substitute for leg work, it should be recognised that there are fundamental physiological differences between the two forms of exercise and there is need to develop new standards of reference of arm work.

We would like to thank Mrs. Caroline Doré for statistical advice and the subjects for their whole hearted co-operation in this study.

Les réponses physiologiques de 8 sujets masculins en bonne santé ont été étudiées durant l'exercice effectué avec 1 ou 2 bras sur un bicycle ergométrique convenablement modifié, dans des conditions soigneusement standardisées, utilisant une planche maintenant les épaules et un harnais destiné à empêcher les mouvements du tronc.

Le rendement mécanique " apparent " (*E*—défini comme le rapport du travail fourni sur la dépense d'énergie aérobie) a été trouvé identique pour des faibles niveaux d'exercice, à celui trouvé antérieurement pour le travail de jambe, environ 0,25; quoi qu'il y ait eu une chute de *E* à des niveaux supérieurs, à environ 40% de la $\dot{V}_{O_2\,max}$. La ventilation minute $\dot{V}_E$ a été supérieure lors d'exercices de bras par rapport à des exercices de jambes pour des $\dot{V}_{O_2}$ ou des $\dot{V}_{CO_2}$ données. Le débit cardiaque $\dot{Q}$ demeure le même pour une $\dot{V}_{O_2}$ donnée, quoique le volume systolique soit diminué et la fréquence cardiaque augmentée lors du travail avec les bras comparativement à celui avec les jambes.

La puissance aérobie maximale des bras a été trouvée égale à 1/3 de celle des jambes, mais, pour un volume donné de membre (muscle + os mesuré d'après la méthode de Jones et Pearson (1969)), elle fut supérieure d'environ 13% pour le travail de bras par rapport à celui des jambes: les raisons de cette différence sont discutées.

Die physiologischen Reaktionen von acht gesunden männlichen Versuchspersonen wurden bei ein- und zweiarmger Kurbelarbeit, die an einem passend verändertem Fahrradergometer unter sorgfältig standardisierten Bedingungen ausgeführt wurde, untersucht. Es wurde dabei die Schulter durch ein Brett zurückgehalten und die Bewegung des Rumpfes durch ein Bandgestell verkleinert. Der reine mechanische Wirkungsgrad (E = definiert als das Verhältnis der geleisteten Arbeit zur aufgewendeten Energie) ähnelte bei leichter Arbeit dem früher bei Beinarbeit gefundenem, nämlich 0,25. E fiel jedoch bei einer Arbeitsschwere oberhalb etwa 40% der maximalen Sauerstoffaufnahme/min. Bei einer gegebenen Sauerstoffaufnahme oder Kohlensäureabgabe war das Atemminutenvolumen bei Armarbeit grösser als bei Beinarbeit. Das Herzminutenvolumen blieb dagegen für eine bestimmte Sauerstoffaufnahme das gleiche, obwohl, verglichen mit Beinarbeit, bei Armarbeit das Schlagvolumen verkleinert und die Herzfrequenz vergrössert wurde. Die maximale aerobe Leistung der Arme war ein Drittel der der Beine. Für ein bestimmtes Beinvolumen jedoch (Muskel + Knochen, gemessen nach der Methode von Jones und Pearson (1969)) war die Leistungsfähigkeit der Arme etwa 13% grösser als die der Beine. Die Gründe dafür werden diskutiert.

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Physiological responses to one- and two-leg exercise breathing air and 45% oxygen

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DAVIES, C. T. M., AND A. J. SARGEANT. *Physiological responses to one- and two-leg exercise breathing air and 45% oxygen.* J. Appl. Physiol. 36(2): 142–148. 1974.—Physiological responses to one- and two-leg exercise breathing air and 45% oxygen have been studied on five healthy male subjects, aged 24–39 yr. The results showed that, although ventilation rate ($\dot{V}_E$) for a given carbon dioxide output ($\dot{V}_{CO_2}$) was marginally increased in one- compared with two-leg exercise, oxygen intake ($\dot{V}_{O_2}$) for given light to moderate work load was identical in both forms of exercise. However, during heavy one-leg exercise there was a small (~5%) decline in "apparent" mechanical efficiency. These relationships were unaffected by an increase in the concentration of inspired oxygen ($F_{I_{O_2}}$). For given $\dot{V}_{O_2}$, cardiac frequency (f_H) was consistently higher in one- than in two-leg exercise, but cardiac output appeared to retain its relationship to $\dot{V}_{O_2}$ irrespective of $F_{I_{O_2}}$ or the type of exercise. The maximum aerobic power of one-leg exercise was 70% of two-leg exercise, but in terms of a given muscle (plus bone) mass the net $\dot{V}_{O_2\,max}$ of a single limb was ~26% higher than when both limbs were used. However, provided a (postural component) correction (~170 ml·min^{-1}) was applied to maximal one-leg work, the difference in $\dot{V}_{O_2\,max}$ of one and two leg(s) could be removed by breathing 45% oxygen. It was suggested that the lower relative $\dot{V}_{O_2\,max}$ of two-leg exercise may be related to limitations imposed by $\dot{Q}$ and the resulting arterial desaturation which may accompany such exercise at maximal levels.

maximum aerobic power; O_2 breathing; muscle mass; mechanical efficiency; cardiac output; ventilation

EXERCISE PERFORMANCE using the legs and/or arms has been studied extensively (see Bevegård and Shepherd (3) for a general review), but work with a single limb (leg or arm) except for the studies of Dunér (12) and Freyschuss and Strandell (13) has been almost wholly neglected. This is very surprising when one thinks of the importance and application of this type of exercise to the fields of rehabilitation physiology and physical medicine.

In the present study we have investigated the physiological responses to one- and two-leg exercise under normoxic and hyperoxic conditions in five healthy male subjects. In addition, estimates of leg (muscle plus bone) volume (LV, 21) were taken in order to explore the relationship between limb composition and exercise performance.

SUBJECTS

The anthropometric details of the five male subjects who took part in the investigation are given in Table 1.

At the time of the investigation the five subjects were normally active but not in training. They had all taken part in physiological investigations, but none of the subjects had performed one-leg exercise before the present study.

METHODS

During the preliminary period, the subjects were allowed to practice one- and two-leg exercise to overcome the confounding influences of learning and habituation to the task (11). In the definitive study the subjects were required to exercise at each of five submaximal work loads using one leg and (on a different occasion) two legs on a stationary bicycle ergometer, while breathing air and 45% oxygen. During one-leg exercise the foot of the active limb was fitted with a plimsoll which was secured to the bicycle pedal by bolts and two metal plates. The bicycle had a fixed wheel and no attempt was made to return the "passive" pedal during cycling (cf. Freyschuss and Strandell (13)).

In submaximal experiments, oxygen intake ($\dot{V}_{O_2}$), minute ventilation ($\dot{V}_E$), and respiratory frequency (f_R) were measured during the 8–10th min of each work load by the standard open-circuit technique (7). Mechanical efficiency (defined as the ratio of work performed to aerobic energy expenditure) was calculated from the $\dot{V}_{O_2}$ work data using a base line of pedaling against zero load (28). In the O_2 breathing experiments gas mixtures were delivered to the subject using a series of rotameters connected in a similar way to that described by Cunningham et al. (6).

On separate occasions maximal performance breathing air and 45% oxygen after a method similar to that described by Binkhorst and Van Leeuwen (4).

Criteria of maximal performance. During two-leg exercise the "$\dot{V}_{O_2}$ plateau" criterion for the attainment of maximal aerobic power ($\dot{V}_{O_2\,max}$) previously developed (7) was

TABLE 1. *Age, weight (wt), height (ht), leg volume (LV), and arm volume (AV) of five subjects*

Subj No.	Age, yr	Wt, kg	Ht, cm	LV, l	AV, l
1	39	63.0	177.3	12.82	3.24
2	28	73.3	179.3	14.40	4.39
3	31	98.0	183.0	17.60	6.47
4	24	77.3	176.5	16.37	4.33
5	26	77.5	184.0	17.43	4.80
Mean	29.6	77.8	180.0	15.72	4.65

applied. For one-leg work it was found that this criterion was not easy to apply. Four of the five subjects did not show a clear plateau of $\dot{V}O_2$ with increasing work at maximal effort due to a small change in "apparent" mechanical efficiency (Fig. 1). To overcome this difficulty we took duplicate measurements of maximal performance on separate occasions. These replicate measurements of $\dot{V}O_2$ were required to agree within ± 5 %. Provided this criterion was met, the mean of these two values were taken as the $\dot{V}O_{2\,max}$ for that exercise; if it was not, the maximal experiments were repeated.

Following the completion of our study, Gleser's (15) paper has been published. In his investigation he appears to have overcome this problem of the change in apparent mechanical efficiency at or near maximal load by arranging for the one-leg work to be performed by pairs of subjects. Each subject was required to stand on a low platform at the side of the bicycle, and pedal the bicycle with the inside leg and use the outside leg as support. This method clearly requires active cooperation from the subjects regarding work sharing, but Gleser's data seem to show that it works. The method, if validated, could form a useful basis for measuring the maximal performance of one-leg exercise in future studies. Nevertheless, it should be noted that Gleser's $\dot{V}O_2$ and $\dot{Q}$ data for one-leg exercise are similar to ours (Tables 2 and 3). His results show that hypoxia reduces

TABLE 2. *Principal indices of exercise performance in five subjects of present investigation*

				A: One-Leg Exercise				
$\dot{W}$, kpm/min	0.21	0	156 ±22	304 ±9	443 ±31	598 ±15	738 ±34	
	0.45	0	147 ±3	301 ±7	450	607 ±30	745 ±55	
$\dot{V}O_2$, l/min STPD	0.21	0.39 ±0.07	0.66 ±0.11	0.91 ±0.08	1.25 ±0.09	1.64 ±0.21	2.06 ±0.15	
	0.45	0.42 ±0.09	0.71 ±0.14	1.01 ±0.06	1.36 ±0.11	1.84 ±0.04	2.24 ±0.22	
$\dot{V}E$, l/min BTPS	0.21	13.4 ±1.7	19.3 ±2.4	26.5 ±2.4	37.5 ±4.0	54.1 ±6.3	73.5 ±15.8	
	0.45	10.2 ±0.9	14.6 ±0.8	23.2 ±1.7	31.1 ±2.0	44.1 ±6.2	58.3 ±7.6	
$\dot{Q}$, l/min	0.21	6.3 ±1.7	8.8 ±1.6	10.6 ±1.1	11.7 ±1.6	14.6 ±2.3	17.4 ±1.1	
	0.45	4.3 ±0.1	7.2 ±1.3	10.3 ±1.6	11.7 ±1.8	14.6 ±1.8	17.7 ±1.6	
f_H, beats/min	0.21	71 ±9	83 ±10	96 ±10	113 ±13	135 ±19	154 ±23	
	0.45	69 ±8	79 ±8	93 ±6	113 ±6	130 ±15	148 ±19	
LA, mg/100 ml	0.21	8.2 ±2.5	7.9 ±1.7	15.5 ±5.4	28.2 ±12.1	40.0 ±20.0	52.3 ±25.0	
	0.45	6.8 ±2.2	8.5 ±2.7	9.2 ±1.3	14.3 ±1.85	22.4 ±3.7	39.9 ±8.8	
				B: Two-Leg Exercise				
$\dot{W}$, kpm/min	0.21	0	120 ±42	287 ±33	456 ±23	619 ±8	905 ±74	1235 ±57	1437 ±107
	0.45	0		293 ±9		597 ±5	892 ±15	1183 ±31	1376 ±65
$\dot{V}O_2$, l/min STPD	0.21	0.44 ±0.10	0.62 ±0.10	0.92 ±0.09	1.20 ±0.02	1.49 ±0.12	2.12 ±0.20	2.71 ±0.05	3.07 ±0.14
	0.45	0.45 ±0.07		0.89 ±0.04		1.40 ±0.13	2.04 ±0.13	2.85 ±0.15	3.19 ±0.01
$\dot{V}E$, l/min BTPS	0.21	15.3 ±3.1	18.2 ±2.6	24.1 ±3.5	33.0 ±4.8	40.2 ±4.5	55.4 ±8.9	86.9 ±5.8	112.0 ±23.2
	0.45	13.4 ±2.0		20.5 ±1.4		32.0 ±1.5	48.4 ±5.3	71.9 ±10.6	101.1 ±12.9
$\dot{Q}$, l/min	0.21	6.05 ±1.34		9.9 ±2.2		13.5 ±1.9	16.2 ±1.4	20.5 ±0.9	21.75 ±1.91
	0.45	7.7 ±1.4		9.9 ±1.8		13.3 ±1.4	15.9 ±0.9	18.6 ±1.3	
f_H, beats/min	0.21	73 ±3	79 ±4	92 ±7	105 ±10	120 ±11	142 ±16	173 ±19	178 ±9
	0.45	71 ±4		87 ±2		111 ±4	139 ±13	167 ±18	172 ±9
LA, mg/100 ml	0.21	7.0 ±1.3		10.6 ±3.3		15.9 ±10.7	20.6 ±8.5	53.5 ±19.9	74.3 ±51
	0.45	7.2 ±1.6		8.6 ±2.8		10.1 ±3.4	17.5 ±8.3	45.7 ±16.0	70.1 ±22

Values are means ± SD. $\dot{W}$ = work output; $\dot{V}O_2$ = oxygen intake; $\dot{V}E$ = minute ventilation; $\dot{Q}$ = cardiac output; f_H = cardiac frequency; LA = blood lactic acid concentration. 1 kpm/min = 0.167 W.

$\dot{V}O_{2\,max}$ and his observations on training support our conclusions concerning the limitations to maximal effort in one- but not two-leg exercise.

Cardiac output. Cardiac output was estimated using a CO_2 rebreathing technique (20), arterial P_{CO_2} being estimated from end-tidal P_{CO_2} after the method suggested by Godfrey and Davies (16). Cardiac frequency was measured using an ECG differential amplifier in conjunction with an instantaneous linear ratemeter. During the final (10th) minute of each exercise load venous blood was sampled from the antecubital vein for subsequent analysis of lactic acid concentration (LA) using the enzymatic method of Gercken (14).

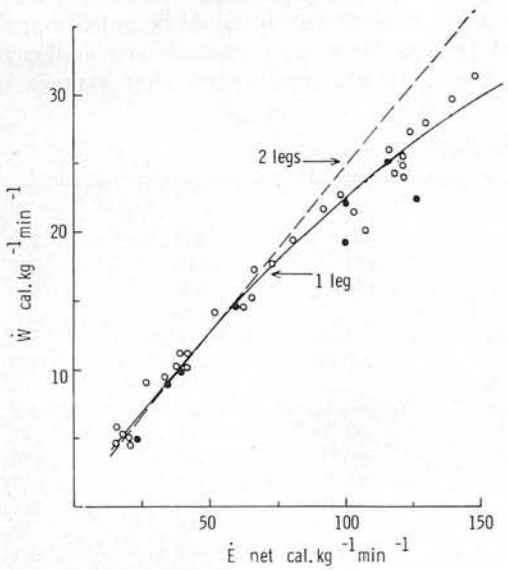

FIG. 1. Relationship of work output to aerobic energy expenditure expressed in cal/kg body wt per min. Symbols: one-leg exercise, $F_{I_{O_2}} = 0.21$ (○) and 0.45 (●). Subjects: *AJS* and *JS*.

RESULTS

The mean values of the principal indices and measurements of exercise performance for one- and two-leg exercise for the five subjects are given in Table 2. The regression equations relating $\dot{V}O_2$, $\dot{V}E$, and f_H and $\dot{Q}$ to their appropriate variable are given in Table 3.

The response of $\dot{V}O_2$ to increasing one- and two-leg exercise was similar in all subjects studied. At the lower work loads $\dot{V}O_2$ in both forms of exercise was identical. However, beyond a work load corresponding to ~700 kpm·min^{-1} (117 W) there was gradual rise in $\dot{V}O_2$ in one-leg work with a corresponding fall in "apparent" mechanical efficiency (see METHODS) from 0.25 to 0.18 (Fig. 1). Breathing oxygen ($F_{I_{O_2}} = 0.45$) was without effect on these relationships.

Minute ventilation ($\dot{V}E$) for a given $\dot{V}CO_2$ was slightly raised in one-leg compared to two-leg exercise (Table 3).

The difference in $\dot{V}E$ for given $\dot{V}CO_2$ of 1.5 l·min^{-1} was of the order of 4.7 l·min^{-1}. Breathing oxygen had little or no effect on $\dot{V}E$ in either one- or two-leg exercise. In three subjects a given $\dot{V}E$ was achieved, at the same VT and f_R regardless of the type of exercise performed and $F_{I_{O_2}}$. In the fourth subject (*JS*), one-leg exercise was achieved with a lower VT and higher f_R than in two-leg work, but again breathing oxygen produced no consistent differences in the $\dot{V}E/\dot{V}T$ relationship.

TABLE 3. *Regression analysis*

	y	*a*	*x*	b_1	b_2	SE_Y	*r*	*n*
			A: Breathing air ($F_{I_{O_2}} = 0.21$)					
1 L	$\dot{V}O_{2\,net}$, l/min	−0.22	Ẇ, kpm/min	0.0025		0.162	0.966	72
2 L		−0.05		0.0018		0.090	0.994	39
1 L	$\dot{V}E_{net}$, ml/kg per min	32.62	$\dot{V}CO_{2\,net}$, ml/kg per min	21.48	0.39	91.91	0.956	74
2 L		3.65		21.55	0.32	53.00	0.985	38
1 L	f_H, beats/min	75.13	$\dot{V}O_{2\,net}$, l/min	45.06		14.83	0.885	74
2 L		72.53		42.85		9.62	0.966	40
1 L	$\dot{Q}$, l/min	4.79	$\dot{V}O_{2\,net}$, l/min	6.08		1.25	0.942	19
2 L		5.03		5.60		1.19	0.954	18
			B: Breathing air enriched with O_2 ($F_{I_{O_2}} = 0.45$)					
1 L	$\dot{V}O_{2\,net}$, l/min	−0.18	Ẇ, kpm/min	0.0025		0.15	0.967	17
2 L		−0.27		0.0022		0.16	0.983	18
1 L	$\dot{V}E_{net}$, ml/kg per min	−31.64	$\dot{V}CO_{2\,net}$, ml/kg per min	33.71	0.03	57.56	0.973	19
2 L		−49.36		30.18	0.03	84.17	0.972	18
1 L	f_H, beats/min	67.64	$\dot{V}O_{2\,net}$, l/min	47.05		12.04	0.921	17
2 L		72.54		39.28		9.54	0.965	18
1 L	$\dot{Q}$, l/min	3.54	$\dot{V}O_{2\,net}$, l/min	6.15		1.33	0.946	15
2 L		6.74		4.27		1.42	0.926	16

Regression analysis of net oxygen intake ($\dot{V}O_{2\,net}$) on work output (Ẇ), net minute ventilation ($\dot{V}E_{net}$) on net carbon dioxide output ($\dot{V}CO_{2\,net}$) expressed in ml/kg body wt per min; cardiac frequency (f_H) on $\dot{V}O_{2\,net}$; cardiac output ($\dot{Q}$) on $\dot{V}O_2$; *A*, breathing air ($F_{I_{O_2}} = 0.21$) and *B*, with the addition of oxygen to the inspired air ($F_{I_{O_2}} = 0.45$). *y* = dependent variable; *x* = independent variable; b_1, and b_2 = regression coefficients of *x*; *a* = constant term; SE_Y = standard error of estimate of *y*; *r* = correlation coefficient; *n* = number.

For a given $\dot{V}O_2$, f_H was consistently higher in one-compared with two-leg work (Table 3). Breathing oxygen produced a fall in f_H in both one- and two-leg exercise but the effect was small. In two-leg work there was a parallel displacement of the $f_H/\dot{V}O_2$ line of approximately 4 beats/min associated with O_2 breathing; in one-leg exercise the effect of O_2 was to decrease the slope and intercept of $f_H/\dot{V}O_2$ curve, resulting in a more marked fall of f_H at the lower work loads. The changes in $\dot{Q}$ were less consistent; in general $\dot{Q}$ retained its relationship to $\dot{V}O_2$ in both forms of exercise regardless of changes in inspired oxygen (Table 3). Thus in all subjects a small fall in cardiac stroke volume (SV) occurred in one-compared with two-leg exercise breathing air but with O_2 breathing no consistent changes of SV were observed.

Blood lactate concentration (LA) was significantly higher a for given $\dot{V}O_2$ in one- compared with two-leg exercise (Table 2) but for given relative work load (expressed as a % $\dot{V}O_{2\ max}$) it was the same in the two forms of exercise. Breathing oxygen produced a fall in LA in both one- and two-leg exercise but since changes in F_{IO_2} were without effect on the $\dot{V}O_{2\ max}$ of one-leg work (Fig. 3), the relationship of LA to % $\dot{V}O_{2\ max}$ was markedly changed (Fig. 2).

Maximal performance. During one-leg maximal exercise, $\dot{V}E$, $\dot{V}O_2$, and f_H reached 80%, 70%, and 94% of their corresponding values for two-leg exercise (Table 4).

Oxygen breathing reduced $\dot{V}E$ in both forms of exercise but was without effect on f_H. Similarly F_{IO_2} had little effect on the $\dot{V}O_{2\ max}$ of one-leg exercise but produced approximately 10% increase in two-leg exercise. The relationship of F_{IO_2} to $\dot{V}O_{2\ max}$ for the two forms of exercise in the present experiments is given in Fig. 3.

Though the differences in the changes of $\dot{V}O_{2\ max}$ with O_2 breathing between subjects were small, there was nevertheless a strong association between the initial value of $\dot{V}O_{2\ max}$ for given subject and the relative change achieved when breathing oxygen (Fig. 4).

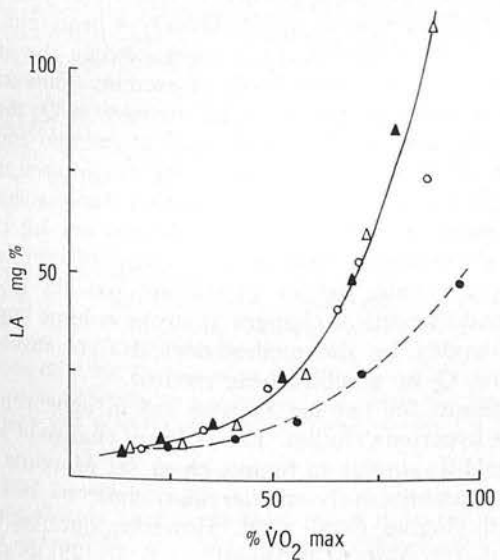

FIG. 2. Blood lactic acid concentration (LA) in relation to relative (expressed as a % $\dot{V}O_{2\ max}$) aerobic energy expenditure. Symbols as for Fig. 1. Response of LA during exercise was similar in 5 subjects measured, data for *subject WP* are shown.

TABLE 4. *Maximum values of absolute and net oxygen intakes, minute ventilation and cardiac frequency during one- and two-leg exercise breathing air and oxygen*

F_{IO_2}	Two Legs				One Leg			
	$\dot{V}E$	$\dot{V}O_2$	f_H	$\dot{V}O_{2\ net}$	$\dot{V}E$	$\dot{V}O_2$	f_H	$\dot{V}O_{2\ net}$
Air, 0.21	153.9 ±19.0	3.50 ±0.42	187 ±5	3.11 ±0.39	121.9 ±22.6	2.41 ±0.34	175 ±13	2.01 ±0.27
O_2, 0.45	147.6 ±18.5	3.83 ±0.46	188 ±7	3.45 ±0.46	106.2 ±19.6	2.38 ±0.21	167 ±14	1.98 ±0.13

Values are means ± SD.

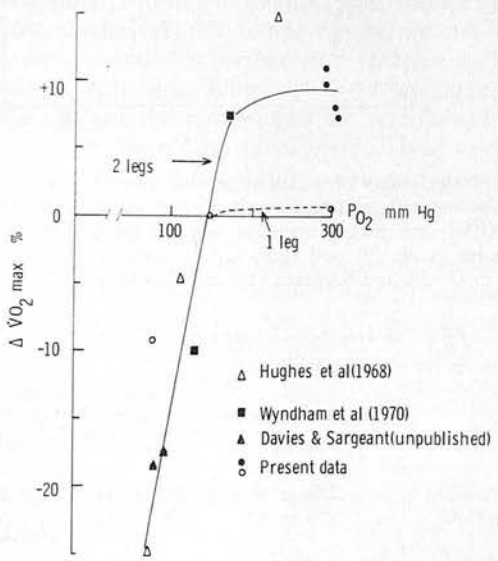

FIG. 3. Relationship of partial pressure of oxygen (P_{O_2}) in the inspired air to % change in maximal aerobic power output (%Δ $\dot{V}O_{2\ max}$).

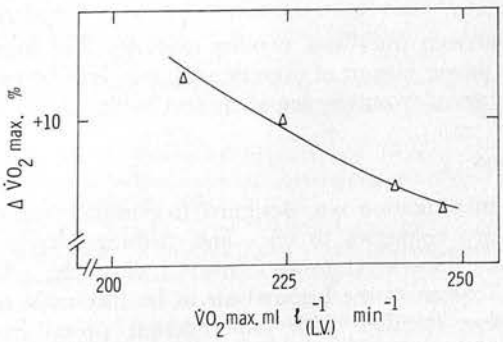

FIG. 4. Relationship of % change of $\dot{V}O_{2\ max}$ to initial $\dot{V}O_{2\ max}$ expressed in ml/l of leg (muscle plus bone) volume per min.

Effective muscle mass. The relationship of $\dot{V}O_{2\ max}$ to estimates of leg (muscle plus bone) volume is shown in Fig. 5.

For a given LV, the net $\dot{V}O_{2\ max}$ of one-leg is approximately 53 $ml \cdot l_{(LV)}^{-1} \cdot min^{-1}$ higher than two-leg exercise. The regression equation relating the two variables is given by:

net $\dot{V}O_{2\ max}$ (l/min) = 0.173 + 0.239 LV (l); r = 0.98

Provided a small correction is made for the positive intercept given in this equation, the effect of breathing O_2 on the net $\dot{V}O_{2\ max}$/LV relationship is to remove the differ-

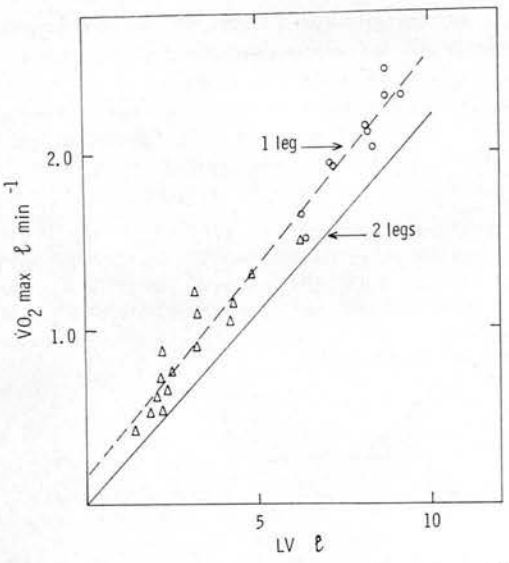

FIG. 5. Relationship of maximal aerobic power output ($\dot{V}O_{2\ max}$) during one-leg exercise (○) to estimates of leg (muscle plus bone) volume (LV)—see text. Regression line for two-leg exercise taken from Davies et al. (9) and data (△) for one- and two-arm work taken from Davies and Sargeant (10) are also shown.

TABLE 5. *Effect of breathing oxygen on net maximum aerobic power output**

$F_{I_{O_2}}$	Net $\dot{V}O_{2\,max}$, ml·l (LV)$^{-1}$·min^{-1}		P
	One leg	Two leg	
Air, 0.21	238 ± 6	205 ± 14	<0.05
O_2, 0.45	236 ± 21	226 ± 11	NS
P	NS	<0.02	

$\dot{V}O_{2\,max}$ is mean ± SD. *Expressed in terms of ml/l of leg muscle plus bone volume (LV) per min during one- and two-leg work.

ences between one- and two-leg exercise. The maximum aerobic power output of exercise with two legs being twice that obtained from one-leg exercise (Table 5).

DISCUSSION

Our investigation was designed to compare the cardiorespiratory responses to one- and two-leg exercise on a stationary bicycle ergometer and to show the effects of adding oxygen to the inspired air in healthy male subjects who were familiar with experimental procedures and thoroughly habituated to cranking exercise. We purposely chose an oxygen enriched inspired gas mixture below an $F_{I_{O_2}}$ of 0.50 to minimize the errors of calculating $\dot{V}O_2$ described by Hill et al. (18). Our findings for two-leg exercise breathing air and 40% oxygen are in general agreement with the work of Bannister and Cunningham (2) and Hughes et al. (19).

For given $\dot{W}$ the $\dot{V}O_2$ is higher in one-leg than in two-leg exercise (Table 3) which confirms the findings of Freyschuss and Strandell (13). The equations given in Table 3 and Freyschuss and Strandell's paper, however, do not describe adequately the curve shown in Fig. 1 due to a curvilinear nature of the relationship between aerobic energy expenditure and $\dot{W}$ in one-leg exercise. The $\dot{W}/\dot{V}O_2$ relationship only changed in one-leg exercise beyond an exercise level corresponding to ~40% $\dot{V}O_{2\,max}$ (Fig. 1). It is unlikely that the mechanical efficiency of the muscle groups of one-leg vary intrinsically from that of the other, when performing cranking movements almost identical in nature.

The most plausible explanation for the discrepancy found in mechanical efficiency between two- and one-leg exercise in the present study is probably to be found in the increased postural component of one-leg exercise as the subject approaches maximum effort. Indeed if the positive intercept of 170 ml/min (Fig. 5) found in the relationship of net $\dot{V}O_{2\,max}$ to LV can be considered as reflecting the postural component and if allowance is made for this, then the difference in calculated apparent mechanical efficiency of one- and two-leg cranking at high levels of exercise is removed. Breathing oxygen had no effect on the relationship of $\dot{V}O_2$ to $\dot{W}$ but it did reduce the LA component in both forms of exercise (Table 3). This supports the view that LA contributes little to the overall energy expenditure during the steady state of exercise (24).

In the present experiments, for a given $\dot{V}O_2$, $\dot{V}E$ is higher in one- compared with two-leg exercise. This finding is consistent with those of Dunér (12), Freyschuss and Strandell (13), and with several other investigators (5, 27) who have studied different forms of exercise involving small muscle masses. However, we feel that exercise $\dot{V}E$ is best described in terms of $\dot{V}CO_2$ rather than at constant $\dot{V}O_2$. If $\dot{V}E$ is related to $\dot{V}CO_2$ the difference between one- and two-leg exercise is small (Table 3) and nonsignificant, and the effect of breathing oxygen in both forms of leg cranking is to produce a small reduction in $\dot{V}E$ for a given $\dot{V}CO_2$ as has been found for other forms of dynamic exercise (1).

For a given work output, f_H is higher in one- than two-leg exercise, consistent with the findings of Freyschuss and Strandell (13). Breathing a gas mixture enriched with oxygen reduces the f_H response in both forms of exercise but the effect in two-leg work is small and results in a parallel displacement of approximately 4 beats/min in the $f_H/\dot{V}O_2$ curve (Table 3). In one-leg exercise the effect of O_2 is greater at the lower levels of exercise. These changes in f_H were not reflected by equal changes in $\dot{Q}$; for given $\dot{V}O_2$, $\dot{Q}$ was similar in the two types of exercise and independent of fractional concentration of inspired oxygen (Table 3). These findings taken together imply a change in cardiac stroke volume between two- and one-leg exercise during air breathing and in both forms of exercise as a result of increasing oxygen in the inspired air. However, these small theoretical changes in stroke volume appear to be confounded by the methodological error involved in measuring $\dot{Q}$ by a rebreathing method.

Our results for two-leg exercise are in agreement with previous hyperoxia studies. The order of change of $\dot{V}O_{2\,max}$ being +11%, similar to figures given by Margaria et al. (23) but quantitatively smaller than those by Hill et al. (18) and Hughes et al. (19). However, our results also suggest that in contrast to Wyndham et al. (29) the change in $\dot{V}O_{2\,max}$ is a curvilinear function of P_{O_2} (Fig. 3), and that it is related to the initial $\dot{V}O_{2\,max}$ of the subject (Fig. 4). Saltin et al. (26) have reported a similar relationship of

$\Delta\dot{V}O_{2\,max}/\dot{V}O_{2\,max}$ during a physical training program. However, for one-leg exercise the $\dot{V}O_{2\,max}$ appears to be independent of F_{IO_2} over the range studied (cf. Gleser (15): during O_2 breathing the $\dot{V}O_{2\,max}$ of a single limb remains unchanged (Table 5).

This suggests that the factors limiting to maximal exercise are different in the two types of exercise. In one-leg cranking, since $\dot{Q}$ for given $\dot{V}O_2$ is the same as two-limb exercise, but the effective muscle mass halved, it would seem reasonable to assume that blood flow to the active limb is greater than two-leg exercise. Indeed, Freyschuss and Strandell (13) report a lower arteriovenous O_2 difference in the active compared with inactive limb in this type of exercise. This is compatible with higher blood flow to active muscle, and under these conditions where $\dot{Q}$ is not limiting (Table 4) one might suppose that the content and saturation of arterial blood with oxygen is maintained. In two-leg exercise the situation is very different. Åstrand and Saltin (1) have shown that the muscle involved in two-leg cycling is adequate and, as exercise is progressively increased, the ability of the heart to increase its output becomes limiting to further aerobic effort (3). Under these conditions Rowell et al. (25) have shown that arterial desaturation of the order of 1–2% occurs. If oxygen is breathed, arterial O_2 may be increased by 1.3 ml/100 ml (3) or 325 ml/min if we assume a $\dot{Q}_{max}$ of 25 $l \cdot min^{-1}$. Thus one might expect an increase in $\dot{V}O_{2\,max}$ of 10% for subjects with mean $\dot{V}O_{2\,max}$ of 3.3 l/min (Table 4). This is exactly the figure we have found in practice for the present experiments (Table 5). Thus one might tentatively suggest that in two-leg exercise the limits imposed by $\dot{Q}$ result in arterial desaturation at maximal effort which can be removed by breathing air enriched with oxygen. In one-leg cycling (Table 4) maximal $\dot{Q}$ is probably not reached. Arterial desaturation is absent and under these conditions the limit to performance is governed by the effective muscle pass per se rather than ability of the heart to increase its output.

These findings and conclusions throw an interesting light on the current debate in the literature (17, 22) concerning human muscle performance. Despite the large body of evidence to contrary (3), several authors (see Kaijser (22) for general review) suggest that the limiting factor to physical performance during rhythmic exercise with the legs is ultimately peripheral in origin and related to O_2 utilization within the muscle. The results from this study show conclusively that in one-leg exercise the working muscles are capable of a further 10% increase in $\dot{V}O_2$ per unit volume over that found in two-leg exercise. This difference can only be removed by the addition of oxygen to the inspired air in the latter case. These results are difficult to reconcile with a theory based purely on peripheral oxygen utilization as the limiting factor to exercise involving large muscle groups.

Finally, the association of $\dot{V}O_{2\,max}$ with leg (muscle plus bone) volume in one-leg exercise (Fig. 5) confirms our previous findings in arm cranking (10), but the effects of breathing oxygen in two-leg exercise in the present study support our view (8) that the relationship between the two variables is not causal: $\dot{V}O_{2\,max}$ can be varied independently of muscle mass.

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Plasma Catecholamine Concentration during Dynamic Exercise Involving Different Muscle Groups

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Abstract. The change in plasma catecholamine concentration (ΔC) has been studied in four healthy male subjects during work, involving different muscle groups, whilst breathing air and 45% oxygen.

The results show that during arm and (1- and 2-) leg(s) work ΔC was more closely associated with relative (expressed as a % of $\dot{V}_{O_2\,max}$) than absolute work load; a rise in C occurred at $\sim 60\%$ $\dot{V}_{O_2\,max}$ in all 3 forms of exercise. However, in arm and 1-leg work the curves relating ΔC to % $\dot{V}_{O_2\,max}$ were displaced to the right indicating the independence of the two variables. Further, breathing 45% oxygen reduced ΔC but was without effect on either $\dot{V}_{O_2}$ at a given work load or $\dot{V}_{O_2\,max}$.

For a given $\dot{V}_{O_2}$, ΔC was inversely related to the effective muscle (plus bone) volume used to perform the work and associated with change of blood lactic acid (LA) concentration, but again the use of exercise involving different muscle groups indicated that the changes in C and LA were essentially independent. This was also true of the changes of C with cardiac output but not cardiac frequency (f_H).

Plasma C changed as a curvilinear function of f_H, the association between the two variables being independent of type of exercise and inspired O_2 concentration within the range used in this study. This suggests that the rise in C and f_H in exercise may be closely related to circulatory stress and may reflect the degree of vasoconstriction present in 'non-active' tissues and efficacy of the body's ability to maintain the integrity of systemic blood pressure in the face of increased demands of the exercising muscles for blood and the transport of oxygen.

Key words: Catecholamines — Muscle Mass — Exercise — Circulation — Maximum Aerobic Power.

There have been several studies of exercise using small muscle groups (see, e.g. Åstrand and Saltin, [4]; Bobbert [9]). These studies show conclusively that during work with small muscle groups, for example, arm compared with leg cranking, there is evidence of greater physiological strain and an increase in the anaerobic component of exercise. During such work there is an enhanced cardiac and ventilatory response [1, 2, 11, 26] and a rise in systemic blood pressure [3]. Freyschuss and Strandell [18] have postulated that during work with small muscle groups there must be an increased sympathetic involvement and a subsequent rise in

Table 1. Physical characteristics of the 4 healthy male subjects: Age, weight (Wt), height (Ht), arm (muscle plus bone) volume (AV), leg (muscle plus bone) volume (LV) and the maximum aerobic power output ($\dot{V}_{O_2\,max}$) for arm, and 1- and 2-leg work

Subject no.	Age (yr.)	Wt (kg)	Ht (cm)	AV	LV	$\dot{V}_{O_2\,max}$ l min^{-1} Arm	1-leg	2-leg
1	39	63.0	177.3	3.24	12.82	1.20	2.01	3.10
2	28	73.3	179.3	4.39	14.40	1.53	2.27	3.21
3	31	98.0	183.0	6.47	17.60	1.97	2.78	3.69
4	24	77.3	176.5	4.33	16.37	1.78	2.59	4.00
Mean	30.5	77.9	179.0	4.61	15.30	1.62	2.41	3.50
SD	± 6.3	±14.7	±2.9	±1.35	± 2.11	±0.33	±0.34	±0.42

plasma catecholamine, but to our knowledge there have been no detailed studies of this problem. Vendsalu [28] and Häggendal *et al.* [20] have reported the changes in plasma catecholamine level (ΔC) during work with the legs but give no data for exercise involving other muscle groups.

We have studied ΔC in four healthy male subjects, doing work with the arms and one- and two leg(s) on suitably modified stationary bicycle ergometer. We also present some observations on the effects of breathing oxygen on plasma catecholamine changes during exercise.

Materials and Methods

The anthropometric details of the four subjects who participated in this investigation are given in Table 1.

Exercise was performed on modified stationary bicycle ergometer which allowed 1-leg or 2-leg or arm work to be carried out on a given occasion. Each subject performed the 3 exercises over a wide range of work intensities up to and including maximal levels. Each work level was performed for 10 min and blood was sampled from the antecubital vein during the final minute and subsequently analysed for lactic acid and catecholamine using the methods of Gercken [19] and McCullough [25] respectively. *Oxygen intake* ($\dot{V}_{O_2}$) and minute ventilation were measured using an open circuit technique over the 7th to 9th min at each work level. Cardiac output was estimated using a CO_2 rebreathing method [21] during the final minute of exercise following the blood collection and cardiac frequency was measured using a differential ECG amplifier in conjunction with a linear instantaneous rate meter. In the O_2 breathing experiments gas mixtures were delivered to the subject using a series of rotameters assembled in a way similar to that described by Cunningham *et al.* [12].

Maximal Oxygen Intake ($\dot{V}_{O_2\,max}$). Maximal aerobic power ($\dot{V}_{O_2\,max}$) breathing air and 45% oxygen was determined for 2-leg, 1-leg and arm work on separate occasions using a continuous work method similar to that described by Binkhorst and Van Leeuwen [8]. For 2-leg work the '$\dot{V}_{O_2}$ plateau' criterion, i.e. $\dot{V}_{O_2}$ shows no further rise with increasing work load: plateau values to agree within ± 5% previously developed [13] was applied. However, during maximal arm and 1-leg work none of the subjects showed a clear plateau when $\dot{V}_{O_2}$ was plotted against

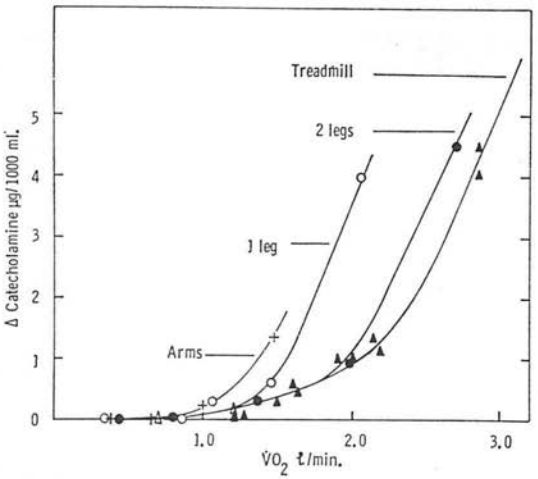

Fig. 1. Changes in plasma catecholamine (ΔC) during arm (+), one-leg (○) and two-leg (●) work on a stationary bicycle ergometer and walking at various gradients at 6.44 km · h^{-1} on a motor driven treadmill (▲). Subject: (2)

work load. To overcome this difficulty we took duplicate measurements of maximal performance, on separate occasions and provided the two $\dot{V}_{O_2}$ values agreed within ± 5% this was taken as evidence of $\dot{V}_{O_2\,max}$ having been reached. If the replicate $\dot{V}_{O_2}$ values did not agree within the required limits, the maximal experiments were repeated. This method of measuring $\dot{V}_{O_2\,max}$ with relatively small muscle groups has been discussed previously [14, 15] and the reader is referred to these publications for further details.

Leg and arm (muscle plus bone) volumes were estimated at the time of the exercise experiments using the method of Jones and Pearson [22].

Results

The mean changes in catecholamine and lactic acid concentrations, oxygen intake, cardiac output and cardiac frequency of the four subjects during the different forms of exercise are given in Table 2.

For a given form of exercise the changes in catecholamine concentration show a similar pattern (Fig. 1). In arm and 1- and 2-leg work at low levels of exercise, there is little or no increase in catecholamine concentration, but as the intensity work increases beyond a certain critical level, a rapid rise in C occurs. The absolute level of $\dot{V}_{O_2}$ at which the change in catecholamine concentration occurs is clearly dependent upon the muscle (plus bone) volume used to perform the work (Fig. 2), and the maximum aerobic power output of the individual subject. If the changes of catecholamine concentration are related to relative (expressed as a % $\dot{V}_{O_2\,max}$) rather than absolute work load, then the individual differences for a

Table 2. Work output ($\dot{W}$) oxygen intake ($\dot{V}_{O_2}$), % maximum aerobic power output (% $\dot{V}_{O_2\,max}$), cardiac frequency (f_H), cardiac output ($\dot{Q}$) and changes in blood lactate (ΔLA) and catecholamine (ΔC) during exercise with the arms and 1- and 2-legs

Subject	$\dot{W}$ kpm min^{-1}	$\dot{V}_{O_2}$ l · min^{-1}	% $\dot{V}_{O_2\,max}$	f_H bts · min^{-1}	$\dot{Q}$ l · min^{-1}	ΔC µg/l	ΔLA mg/100 ml	$\dot{W}$ kpm min^{-1}	$\dot{V}_{O_2}$ l · min^{-1}	% $\dot{V}_{O_2\,max}$	f_H bts · min^{-1}	$\dot{Q}$ l · min^{-1}	ΔC µg/l	ΔLA mg/100 ml
	1-leg exercise F_{IO_2} = 0.21							2-leg exercise F_{IO_2} = 0.21						
1	0	0.31	15	75	5.3	0	0	0	0.37	11	74	5.1	0	0
	145	0.56	28	81	7.0	0.45	−1.8	300	0.86	28	91	8.1	0.15	2.2
	289	0.81	40	95	9.6	0.45	0.2	622	1.47	47	129	11.8	0.98	1.4
	434	1.16	57	112	9.9	0.98	7.5	932	2.04	66	158	14.4	2.03	10.4
	590	1.63	80	148	13.2	1.80	25.7	1296	2.67	86	175	20.2	5.83	38.2
	780	2.02	100	175	16.3	5.18	65.7							
2	0	0.35	15	65	5.3	0	0	0	0.39	9	73	7.0	0	0
	300	0.84	37	93	9.8	0.08	5.4	287	0.83	26	93	7.9	−0.05	2.0
	450	1.10	48	125	11.1	0.38	15.5	622	1.34	42	121	12.0	0.37	8.8
	600	1.48	65	152	14.0	0.68	35.6	900	2.00	62	158	15.5	1.05	25.4
	720	2.10	92	185	17.8	4.00	59.1	1242	2.71	85	193	21.0	4.57	70.3
3	0	0.44	16	79	8.2	0	0	0	0.56	15	77	8.1	0	0
	325	0.95	34	100	11.2	0.22	6.4	300	0.99	27	93	11.7	0.30	1.9
	450	1.35	49	116	13.8	0.37	17.0	623	1.56	42	126	15.4	0.45	6.6
	624	1.81	65	146	18.0	1.50	34.2	900	2.11	57	151	18.2	0.98	18.2
	750	1.95	70	162	18.8	2.40	46.1	1158	2.68	73	173	21.4	2.77	52.8
	860	2.44	88	175	—	4.85	65.3	1560	3.23	88	186	23.1	5.77	102.5
4	0	0.48	18	75	—	0	0	0	0.50	13	70	—	0	0
	150	0.62	24	83	9.7	0	0	311	0.98	25	87	11.9	−0.08	1.2
	300	0.91	35	95	11.9	0.45	2.2	622	1.37	34	108	15.0	−0.08	2.2
	600	1.03	40	112	—	0.60	12.9	900	2.00	50	125	16.8	0.37	11.3
	750	1.99	77	128	16.9	1.20	22.1	1244	2.79	70	148	19.4	1.27	23.5
	865	2.44	91	151	18.3	3.20	53.0	1365	2.96	74	170	20.4	3.00	32.6

	1-leg exercise $F_{IO_2} = 0.45$							2-leg exercise $F_{IO_2} = 0.45$						
1	0	0.33	15	60	4.4	0	0	0	0.41	12	67	6.0	0	0
	150	0.60	28	74	6.3	—0.15	3.2	300	0.88	26	85	8.0	0	−0.8
	300	0.94	44	87	9.2	—0.08	−0.3	600	1.43	43	112	11.5	0.15	0.3
	450	1.34	63	109	10.0	0.15	4.1	900	1.96	58	143	14.7	0.25	3.5
	600	1.80	85	135	12.2	0.97	15.6	1220	2.81	84	167	17.0	3.53	24.8
	750	1.94	91	157	—	1.95	37.8							
2	0	0.42	18	73	4.2	0	0	0	0.41	12	70	8.0	0	0
	294	0.98	43	95	8.7	0.23	5.6	283	0.91	26	87	10.0	−0.05	0.5
	450	1.26	55	110	11.5	—	11.8	590	1.52	43	111	12.7	0.30	7.1
	580	1.85	81	141	15.7	—	17.9	870	2.17	62	147	16.3	0.45	19.3
	662	2.29	100	162	18.2	1.20	39.3	1160	3.05	87	185	20.0	2.30	60.6
3	0	0.45	16	80	—	0	0	0	0.56	14	76	7.4	0	0
	144	0.66	25	89	8.2	0	0	288	0.94	23	88	9.8	−0.05	3.7
	300	1.08	41	100	11.6	0	4.3	600	1.43	35	115	14.5	0.50	6.6
	450	1.47	56	120	13.6	0.23	8.1	900	2.14	52	147	16.7	0.75	17.9
	600	1.87	72	135	16.1	0.68	20.0	1155	2.86	69	174	18.2	2.30	42.9
	750	2.49	95	165	19.7	1.73	40.9	1330	3.20	80	179	—	4.65	78.8
	1040	2.78	100	180	—	4.80	57.1							
4	0	0.51	19	75	—	0	0	0	0.44	10	70	9.4	0	0
	148	0.86	32	75	—	−0.15	0	300	0.85	20	91	11.7	0.16	−1.1
	312	1.03	39	89	11.7	0.15	0.6	600	1.22	29	106	14.4	0.26	−2.5
	650	1.50[a]	56	108	14.4	0.52	9.8	900	1.91	44	119	16.0	0.66	0.3
	750	2.09	78	124	16.0	0.80	18.2	1200	2.70	63	143	19.1	1.15	22.7
	815	2.38	90	131	16.9	1.50	29.1	1422	3.19	75	166	—	3.00	45.3

[a] Predicted from work output.

Table 2 (continued)

Arm exercise $F_{IO_2} = 0.21$							
Subject	$\dot{W}$ kpm min^{-1}	$\dot{V}_{O_2}$ l · min^{-1}	% $\dot{V}_{O_2\,max}$	f_H bts · min^{-1}	$\dot{Q}$ l · min^{-1}	ΔC µg/l	ΔLA mg/100 ml
1	0	0.32	27	66	—	0	0
	75	0.40	33	72	—	0.15	2.0
	150	0.62	52	88	—	0.25	29.1
	230	0.80	67	108	—	0.45	45.3
	311	1.04	87	143	—	1.58	79.9
2	0	0.37	24	63	—	0	0
	155	0.66	43	85	—	−0.13	9.3
	300	0.99	65	113	—	0.28	21.5
	450	1.50[a]	98	175	—	1.33	57.4

[a] Predicted from work output.

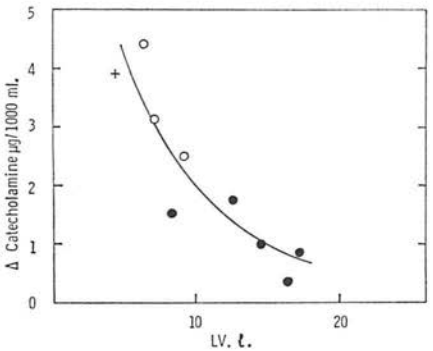

Fig. 2. Changes in plasma catecholamine (ΔC) at a given $\dot{V}_{O_2}$ of 2 l · min^{-1} in relation to estimates of limb (muscle plus bone) volume (LV) using the physical anthropometric method of Jones and Pearson [22]. Symbols as for Fig. 1

given exercise disappear (Fig. 3), but the shape of the curves for 1-leg, 2-leg and arm work are different (Fig. 3). Thus, for a given exercise, although ΔC and % $\dot{V}_{O_2\,max}$ are closely associated, the two variables are essentially independent. The independence of ΔC and % $\dot{V}_{O_2\,max}$ is further revealed by the effect of O_2 breathing during 1- and 2-leg work. For a given $\dot{V}_{O_2}$ the addition of oxygen to the inspired air reduced ΔC in both forms of exercise but whereas in 2-leg exercise there is an associated increase in $\dot{V}_{O_2\,max}$ in 1-leg work this is not seen, thus ΔC and $\dot{V}_{O_2\,max}$ change independently (Fig. 4).

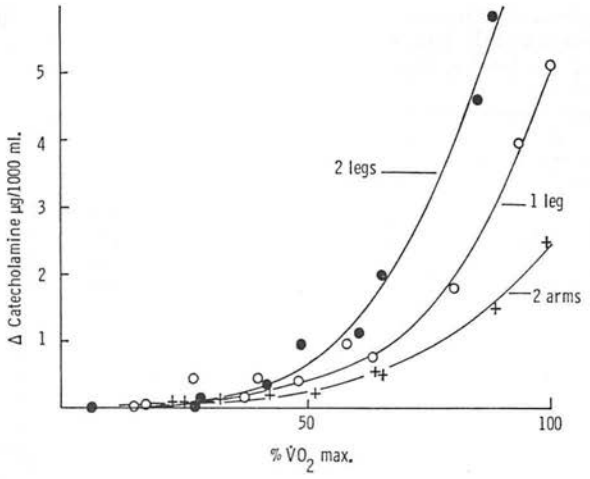

Fig. 3. Relationship of changes in catecholamine to relative aerobic work output (expressed as a % of $\dot{V}_{O_2\,max}$). Symbols as for Fig. 1. Subjects: (1) and (2)

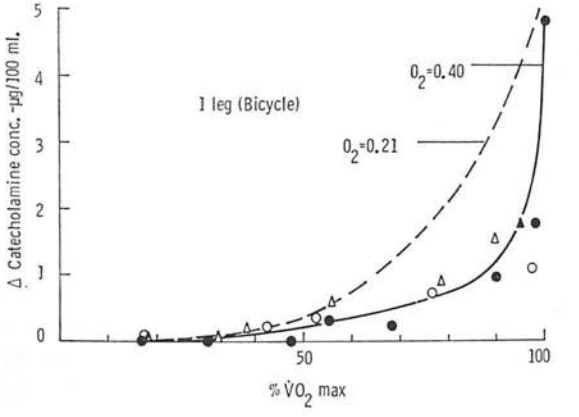

Fig. 4. The effect of breathing air enriched with oxygen ($F_{IO_2} = 0.45$) on changes of plasma catecholamine (ΔC) during one leg exercise. $F_{IO_2} = 0.21$ ----; $F_{IO_2} = 0.45$ ———. Subjects (1) (●); (2) (○) (3) ▲; and (4) (△)

The same holds true if one considers blood lactic acid concentration. For a given exercise there is a close association between ΔC and LA (Table 3), but the relationship is dependent on the type of exercise performed.

Table 3. Regression analysis of ΔC on ΔLA and $\log_{10}(\Delta C + 1)$ on % $\dot{V}_{O_2\,max}$, $\dot{Q}$ and f_H. The equation is of the form $y = a + bx$. r = correlation coefficient, n = degrees of freedom and P = level of significance of the comparison between the regression equations in the different forms of work

Exercise	y	a	x	b	r	n	P
Arm (0.21)		−0.117		0.0209	0.93	9	
1 leg (0.21)		−0.085		0.0649	0.94	22	0.05
2 leg (0.21)	ΔC	0.018	ΔLA	0.0622	0.88	22	
1 leg (0.45)		−0.161		0.0610	0.92	21	
2 leg (0.45)		0.120		0.0536	0.90	22	
Arm (0.21)		−0.183		0.0056	0.90	9	
1 leg (0.21)		−0.194		0.0090	0.94	22	
2 leg (0.21)	$\log \Delta C + 1$	−0.230	% $\dot{V}_{O_2\,max}$	0.0105	0.94	22	0.05
1 leg (0.45)		−0.210		0.0066	0.89	21	
2 leg (0.45)		−0.174		0.0087	0.89	22	
1 leg (0.21)		−0.312		0.0466	0.82	20	
2 leg (0.21)	$\log \Delta C + 1$	−0.383	$\dot{Q}$	0.0456	0.84	21	0.05
1 leg (0.45)		−0.215		0.0297	0.87	17	
2 leg (0.45)		−0.356		0.0396	0.83	20	
Arm (0.21)		−0.268		0.0039	0.90	9	
1 leg (0.21)	$\log \Delta C + 1$	−0.506	f_H	0.0065	0.92	22	NS
2 leg (0.21)		−0.586		0.0067	0.93	22	
1 leg (0.45)		−0.483		0.0058	0.92	21	
2 leg (0.45)		−0.472		0.0056	0.90	22	

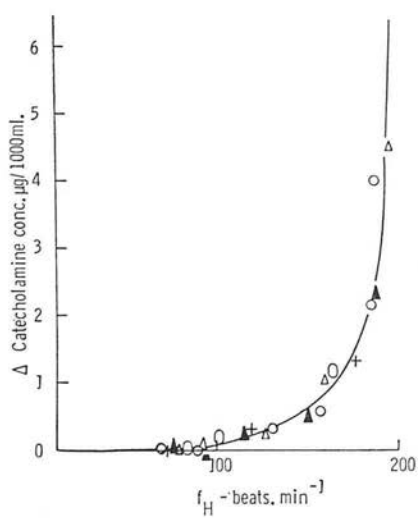

Fig. 5. The relationship of changes of plasma catecholamine (ΔC) to exercise cardiac frequency (f_H). Symbols: 2 legs, $F_{I_{O_2}} = 0.21$ (△); 2 legs $F_{I_{O_2}} = 0.45$ (▲); 1 leg $F_{I_{O_2}} = 0.21$ (○); 1 leg $F_{I_{O_2}} = 0.45$ (●); Arms $F_{I_{O_2}} = 0.21$ (+). Subject (2)

For a given subject there is also a close association of $\dot{Q}$ and ΔC but again for different forms of exercise the two variables again appear to be independent (Table 3). This is not so for cardiac frequency, the change of plasma catecholamine concentration is closely associated with f_H irrespective of the type of exercise performed and fractional concentration of oxygen in the inspired air (Fig. 5 and Table 3). However, there is small but significant inter-subject difference in the relationship of ΔC to f_H. The slope of the individual regression equations are identical, but the intercept of the line $\log_{10} \Delta C$ on f_H on the Y axis, however, varies significantly ($P < 0.001$) between subjects.

Discussion

Since the introduction of sensitive biochemical methods for the estimation of physiological changes in plasma catecholamines [24] several authors [11, 23, 28] have noted that heavy exercise is associated with a rise in C. Our results confirm their findings and show, in agreement with Häggendal *et al.* [20], that for a given form of exercise ΔC is more closely related to relative (expressed as % $\dot{V}_{O_2\,max}$) than absolute aerobic energy expenditure (Fig. 3). The major difference between Häggendal *et al.*'s experimental results and our own is that in cycling they found the relative work threshold beyond which ΔC began to increase occurred close to the maximum aerobic power output of the subject. In the present study the marked rise in the ΔC/% $\dot{V}_{O_2\,max}$ curve occurred at $\sim 60\%$ $\dot{V}_{O_2\,max}$ in all 3 types of work investigated. The difference in experimental results may in part be due to the different type of subjects and exercise procedures used in the two studies. We used sedentary subjects and a continuous exercise procedure in which the work level was increased every 10 min, venous blood being sampled during the final minute at each load whereas two of the five subjects in Häggendal *et al.*'s [20] investigation were relatively well-trained, discontinuous exercise was given and arterial blood sampling was used. Since Vendsalu [28] has shown that the total catecholamine concentration of venous blood is higher than arterial blood it would seem reasonable to expect ΔC for given $\dot{V}_{O_2}$ to be lower and the level of % $\dot{V}_{O_2\,max}$ beyond which ΔC shows a marked rise to be correspondingly raised in Häggendal and his co-workers' study compared with our own.

The rise in C during exercise is dependent on the *effective* limb (muscle plus bone) volume used to perform the exercise (Fig. 2) and the fractional concentration of oxygen in the inspired air (Fig. 4). The measured C for given $\dot{V}_{O_2}$ in arm work (Fig. 2) gives support to the view that there is a greater sympathetic involvement in exercise involving small muscle groups [6, 18]. However, the relationship of ΔC to the muscle mass used to perform the work in the present investigation, is

non-linear (Fig. 2). This finding taken together with relationship of ΔC to LA given in Table 3 suggests that the rise in C during exercise is associated with peripheral (muscle) events but is not dependent upon them.

The closest association of ΔC, to be found in this study, is with central circulatory factors particularly exercise cardiac frequency (Fig. 5). The relationship of ΔC to f_H explains from 72% to 88% of the total variance of the two variables in the individual case (Table 3), and suggests that the rise in C may be implicated in the changes of exercise f_H which occur irrespective of the type of dynamic work performed, and fractional concentration of inspired oxygen. However, the reasons underlying this association of ΔC with f_H we have found are less clear.

The data of Vendsalu [28] suggest that the major change in C in venous blood during exercise is due to nor-adrenaline. The only known functions of nor-adrenaline are to produce blood vessel vasoconstriction and to stimulate cardiac function via β receptors situated in the heart. Therefore one might speculate on the basis of our results that during dynamic exercise with the legs, the rise in ΔC and f_H reflects the degree of vasoconstriction (primarily in 'non-active' areas — vasoconstrictor tone in working muscle is known to be overcome by local factors [5]) and the body's attempt to maintain cardiac output and sustain systemic blood pressure in the face of the circulatory stress imposed by the working muscles demand for increased blood flow as exercise progresses beyond a certain critical threshold which in our experiments correspond to $\sim 60\%$ $\dot{V}_{O_2\,max}$. In arm work which utilizes a relatively small 'active' muscle mass the degree of vasoconstriction in the 'inactive' areas in absolute terms might be expected to be greater, because of the necessity of trying to maintain an adequate venous return and cardiac output in this type of exercise [7]. From this viewpoint, one would expect a rise in C for a given $\dot{V}_{O_2}$ as has been found (Fig. 2) and a concomitant rise in f_H. In other words, consistent with our findings, one is suggesting that dynamic exercise produces an increase in vasoconstrictor tone which for given $\dot{V}_{O_2}$ is inversely related to the amount of effective muscle mass used to perform the work and directly associated with efficacy of the cardiac response as indicated by the rise in f_H and the change in cardiac stroke volume.

It is important to realize that the theory does not require a causal link between ΔC and f_H, indeed they are envisaged as associated but independent phenomena. This would seem reasonable from the pharmacological studies of Taylor and Donald [27], and Epstein *et al.* [17] and the cardiac dennervation experiments of Donald and Shepherd [16]. These investigations show that neither the β cell receptor system nor myocardial sympathetic nervous system are essential for eliciting an increase in cardiac frequency or output during work. Indeed, we

see the concomitant changes of exercise ΔC and f_H as two links of a complex chain involved in the control of the central circulation and aortic blood pressure during work. The rise in f_H during work being elicited by the baro-receptors and mediated by the sympathetio nervous system and the release of nor-adrenaline. During the steady state of exercise it is probably as suggested by Bevegård and Shepherd [7], that stroke volume is so regulated as to keep $\dot{Q}$ and aortic blood pressure fairly constant. Thus, during rhythmic dynamic exercise involving different muscle masses, the rise in C and f_H will essentially reflect the degree of sympathetic nor-adrenergic tone and circulatory adjustment, both factors being inextricably linked in the control and maintenance of systemic blood pressure during exercise.

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Indirect Determination of Maximal Aerobic Power Output during Work with One or Two Limbs

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Abstract. The cardiac frequency (f_H) and oxygen intake ($\dot{V}_{O_2}$) responses to submaximal and maximal work with 1- and 2-arms and 1- and 2-legs on suitably modified bicycle ergometers in relation to the prediction of maximal aerobic power output ($\dot{V}_{O_2\,max}$) have been examined in 12 healthy male subjects.

The results showed that the physiological responses to the different forms of submaximal and maximal exercise were distinct and dependent upon the effective muscle mass used to perform the work. The observed $\dot{V}_{O_2\,max}$ of 1-limb could be converted to respective 2-limb value with a coefficient of variation ranging from 4 to 7 %, but maximal work with the arms gave no guide to that of the legs. Extrapolation of the $\dot{V}_{O_2}/f_H$ curve to the $f_{H\,max}$ in 1-leg (175 beats/min) and 2-arm (165 beats/min) resulted in an overestimation of $\dot{V}_{O_2\,max}$ of $+ 70 \pm 200$ ml · min^{-1} and $+ 70 \pm 240$ ml · min^{-1}; but in 1-arm work (153 beats/min), $\dot{V}_{O_2\,max}$ was underestimated by $- 70 \pm 270$ ml · min^{-1}. The bias in predictions for the 3 forms of exercise could be removed by applying the appropriate regression equations relating predicted to observed $\dot{V}_{O_2\,max}$, but the overall accuracy of the extrapolation method was limited to ± 8 %, ± 15 % and ± 23 % in 1-leg, 2-arm and 1-arm work respectively.

It was concluded that maximal work with the upper and lower limbs should be treated separately and if an accuracy of greater than ± 8 to ± 23 % is required in situations where through injury, two limb exercise cannot be performed, attempts should be made to ascertain the $\dot{V}_{O_2\,max}$ of a single limb directly.

Key words: Arm Work — Leg Work — $\dot{V}_{O_2\,max}$ — Prediction — Single Limb — Aerobic Power.

There have been many studies of the $\dot{V}_{O_2\,max}$ of work involving the legs (see Åstrand and Rodahl [4] for general review) but less attention has been given to maximal work either with the arms [1, 5, 7] or a single limb [11, 13, 17]. This is surprising when one considers the importance of these latter types of exercise to the fields of rehabilitation and physical medicine, where it is often difficult, if not impossible, to measure work with the legs in patients who have sustained injury to one (and sometimes both) lower limb(s).

In the present study we have investigated the responses to exercise of 12 healthy male subjects during 1-arm, 1-leg, 2-arm and 2-leg work to see (i) how far existing 2 limb procedures for measuring $\dot{V}_{O_2max}$ directly [2, 8, 18, 19] and indirectly [3, 15, 16] can be applied to exercise with a single limb and (ii) whether work with the arms may be used as a substitute for work with the legs in patients with lower limb injuries.

Material and Methods

Twelve healthy male subjects aged 20 to 42 years were investigated. Their mean physical characteristics were as follows: age (yr): 30.5 ± 6.2; height (cm): 178.2 ± 5.1; weight (kg): 75.1 ± 10.1; arm (muscle plus bone) volume (l): 4.4 ± 0.9, and leg (muscle plus bone) volume (l): 15.5 ± 1.9.

At the time of measurement, all except 4 subjects were accustomed to physiological investigations. Nevertheless, all subjects were required to practise and become familiar with the involved procedures of 1- and 2-limb work on at least one occasion before definitive measurements were taken in order to avoid the confounding influences of learning and habituation to the task [10].

The subjects were required to attend the laboratory on several different occasions either following a light breakfast or lunch and they were asked to refrain from drinking coffee or alcohol prior to the experiment. Four subjects performed 1-leg (right and left) 2-leg, 1-arm (right and left) and 2-arm work; two subjects performed 1- and 2-leg and 2-arm work; two subjects performed 2-leg and 2-arm work; one subject, 1-leg and 2-arm work and one subject 1- and 2-arm work, each form of work being performed at submaximal and maximal effort. Each maximal experiment was repeated twice making a total of 126 separate work sessions for the 12 subjects. All submaximal tests were in the form of continuous exercise and were performed on a suitably modified bicycle ergometer [9]. The work load was raised at 5 min intervals. At least 5 work intensities were covered starting from a baseline of zero load; the aim being to span the full range of a subject's work capacity for a given exercise. During the final minute of each work load, oxygen intake and cardiac frequency were measured using standard techniques previously described [8]. During the different forms of work, care was taken to standardise the position of the subject in relation to the bicycle ergometer. This was particularly true during upper limb exercise where a harness and support board was used to prevent upper body movement and to ensure that the work was performed by the arms alone. The cranking was performed through 360° and involved full extension of the arms; the length of the crank arm was 16.5 cm. This standardisation technique has previously been described in detail [9].

Maximum aerobic power output was determined as a separate experiment using an open circuit technique modified from that described by Binkhorst and van Leeuwen [6].

Results

In order to establish whether the criterion of $\dot{V}_{O_2}$ plateauing which has been used previously in 2-leg work could be applied to single limb exercise, preliminary experiments were formed to establish the relationship of $\dot{V}_{O_2}$ to $\dot{W}$ for 1-leg and arm work. The results are summarised in Fig. 1. The data show that for single limb and arm exercise from a restrained position, the relationship of $\dot{V}_{O_2}$ to $\dot{W}$ is non-linear, but there is no

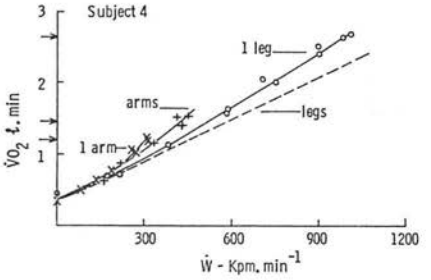

Fig. 1. The relationship of oxygen intake ($\dot{V}_{O_2}$) to work output ($\dot{W}$) for 1-leg (○) 1-(×) and 2-(+) arm work. The arrows on the Y axis indicate the observed maximal aerobic power output ($\dot{V}_{O_2\,max}$) values

Table 1. Maximal responses to exercise and the relationship of observed to predicted $\dot{V}_{O_2\,max}$ for 2-leg, 1-leg, 2-arm and 1-arm work (mean ± SD). Pulmonary ventilation ($\dot{V}_{E\,BTPS}$), oxygen intake ($\dot{V}_{O_2\,max}$) and cardiac frequency ($f_{H\,max}$)

Type of exercise	$\dot{V}_{E\,BTPS}$ (l/min)	$f_{H\,max}$ (beats/min)	$\dot{V}_{O_2\,max}$ (l/min)		Predicted less observed
			Observed	Predicted	
1-arm	48.83	133	1.16	1.09	− 0.07
	± 15.92	± 23	± 0.31	± 0.27	± 0.27
2-arm	74.31	166	1.60	1.67	+ 0.07
	± 19.81	± 12	± 0.28	± 0.36	± 0.24
1-leg	115.81	176	2.58	2.66	+ 0.07
	± 21.56	± 11	± 0.30	± 0.88	± 0.20
2-leg	152.77	191	3.50	3.08	− 0.42
	± 15.35	± 5	± 0.37	± 0.44	± 0.18

tendency for the $\dot{V}_{O_2}/\dot{W}$ to reach an asymptote. Indeed, at maximal work with the subject completely exhausted, the $\dot{V}_{O_2}$ of 1-leg and arm work tended to show a slight upward swing when plotted against $\dot{W}$. This phenomenon presented difficulties in establishing the 'true' $\dot{V}_{O_2\,max}$ of the different types of work undertaken in this investigation. The problem was overcome, by taking duplicate measurements of maximal performance in 1- and 2-arm and 1-leg work. Replicate measurements of $\dot{V}_{O_2}$ at maximal effort were required to agree within ± 5%. Provided this criterion was met, the mean of the two highest recorded values of $\dot{V}_{O_2}$ was taken as the 'true' $\dot{V}_{O_2\,max}$ for that exercise (Fig. 1). The maximal values for minute pulmonary ventilation ($\dot{V}_{E\,max}$), cardiac frequency ($f_{H\,max}$) and oxygen intake ($\dot{V}_{O_2\,max}$) are shown in Table 1 for 1- and 2-leg work and 1- and 2-arm work. During 1-leg and arm work there is a progressive fall in all three parameters from the values found in 2-leg work in

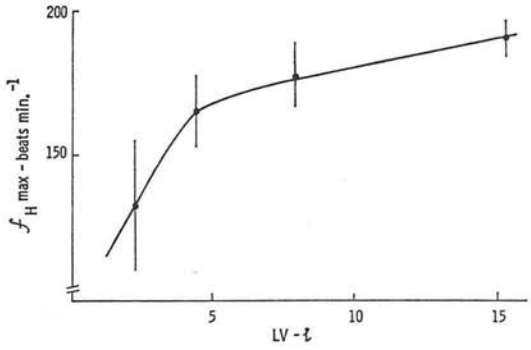

Fig. 2

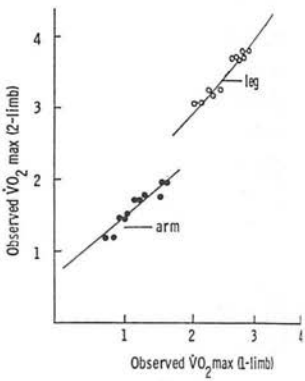

Fig. 3

Fig. 2. The relationship of maximal cardiac frequency ($f_{H\ max}$) to limb (muscle plus bone) volume estimated after the method of Jones and Pearson [14]. It should be noted that the mean age of our subjects is 30 yr. (range 24 to 39 yr). It has been shown previously for 2-leg work [18] that the $f_{H\ max}$ falls by $\sim$ 8 beats/min per decade. This effect of age may have had some bearing on the $f_{H\ max}$ attainable for the various forms of work in the present study and should be borne in mind by the reader when interpreting our data

Fig. 3. Observed maximal aerobic output ($\dot{V}_{O_2\ max}$) with 1 and 2 limbs (○) leg(s) and (●) arm(s). The regression equations relating the $\dot{V}_{O_2\ max}$ of 1-leg to 2-leg and 1-arm to 2-arm are given by: (i) $\dot{V}_{O_2\ max}$ (2 legs) = 1.01 + 0.96 ($\dot{V}_{O_2\ max}$ 1 leg), (l/min) r = 0.90, SD = ± 44 ml. (ii) $\dot{V}_{O_2\ max}$ (2 arms) = 0.72 + 0.79 ($\dot{V}_{O_2\ max}$ 1 arm), r = 0.91, SD = ± 120 ml. The corresponding equation if the $\dot{V}_{O_2\ max}$ of the upper (arm) limbs is related to the lower (leg) limbs is (iii) $\dot{V}_{O_2\ max}$ (2 legs) = 2.62 + 0.56 ($\dot{V}_{O_2\ max}$ 2 arms), r = 0.43, SD = ± 407 ml

association with the effective muscle mass used to perform the work (Fig. 2). The $\dot{V}_{E\ max}$ and $\dot{V}_{O_2\ max}$ for example, of 1-arm work is approximately 30% of that found for 2-leg work and the $f_{H\ max}$ is reduced by some 57 beats/min.

The relationship of 1-limb to 2-limb $\dot{V}_{O_2\ max}$ for the arms and legs is given in Fig. 3.

Submaximal Work

Relationship of f_H to $\dot{V}_{O_2}$. Fig. 4 shows that a linear relationship of $\dot{V}_{O_2}$ to f_H does exist over the greater part of the operative range during submaximal work. However, the $\dot{V}_{O_2}/f_H$ curves for 2-leg, 1-leg and arm work are quite distinct and only overlap at very low levels of exercise. At near maximal levels the behaviour of $\dot{V}_{O_2}/f_H$ for 2-leg work differs markedly from the remaining 3 forms of exercise. In 2-leg work, $\dot{V}_{O_2}$ approaches an asymptote of f_H, whereas in 1-leg and arm work, the linearity of the $\dot{V}_{O_2}/f_H$ curve is maintained (Fig. 4).

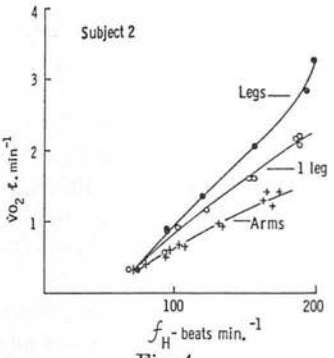

Fig. 4

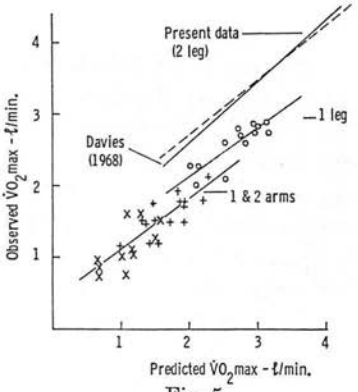

Fig. 5

Fig. 4. The relationship of oxygen intake ($\dot{V}_{O_2}$) to cardiac frequency (f_H) for arm (+); 1-leg (○) and 2-leg (●) work

Fig. 5. The relationship of observed $\dot{V}_{O_2\,max}$ to predicted $\dot{V}_{O_2\,max}$ for leg (○) and 1-(×) and 2-(+) arm work. The solid (heavy) black line is taken from Davies [8] for 2-leg work. The dotted ------ (heavy) black line is for 2-leg work in the present investigation. The regression equations relating observed $\dot{V}_{O_2\,max}$ (y) to predicted $\dot{V}_{O_2\,max}$ (x) for 1-leg and arm work are of the form: $Y = 0.67x + 0.79$ (1-leg work) and $Y = 0.71\,x + 0.41$ (arm work)

In order to assess the intrasubject variability of the $\dot{V}_{O_2}/f_H$ relationship repeated measurements of $\dot{V}_{O_2}$ and f_H were taken during 1-leg exercise in two subjects over a 3 week period. The standard errors of estimate for the regression line and for any new observation were found to be of the order of 130 ml · min^{-1} and 473 ml · min^{-1} respectively.

Prediction of $\dot{V}_{O_2\,max}$. The data given in Eq. (iii) (Fig. 3) and Fig 4 suggest that work with the arms is not indicative of the legs and that for 1- and 2-limb exercise the $\dot{V}_{O_2}/f_H$ curves and $f_{H\,max}$ attainable are specific and therefore the different forms of work for predictive purposes should be treated separately and independently. The data were analysed with this point in mind and presented in Fig. 5 and Table 1. The maximal cardiac frequency values for extrapolation purposes were taken from the smoothed curve given in Fig. 2. For 1-arm, 2-arm, 1-leg and 2-leg exercise these values were 135, 165, 175 and 195 beats/min respectively. As expected (cf. Davies [8]), estimation of $\dot{V}_{O_2\,max}$ from submaximal $\dot{V}_{O_2}/f_H$ values in 2-leg work produces a significant ($P < 0.001$) bias towards underestimation of the 'true' value of the order of — 400 ml/min (Table 1) but for 1-leg work the position is reversed. Extrapolation of the $\dot{V}_{O_2}/f_H$ curve to $f_{H\,max}$ of 175 beats/min produces a small overestimation of $\dot{V}_{O_2\,max}$ of the order of + 70 ± 200 ml/min. 1- and 2-arm work produces an equally realistic prediction of $\dot{V}_{O_2\,max}$ as given for 1-leg exercise but the inter-subject variability is relatively greater.

Discussion

The results of this investigation show that the maximum aerobic power ($\dot{V}_{O_2\,max}$) of a single limb may be measured directly and converted to that of 2 limbs without material loss of accuracy, provided the upper and lower limbs are treated separately. In absolute terms, the observed $\dot{V}_{O_2\,max}$ of the arms is 46% of the legs; the corresponding figure for maximal exercise with 1-leg is 73% (Table 1). However, these differences in $\dot{V}_{O_2\,max}$ are in part influenced by the basal $\dot{V}_{O_2}$ and changes in the postural component of work which may accompany maximal effort in exercise where small muscle masses are involved (see Fig. 1 and Davies and Sargeant [9]). If correction is made for the "basal" $\dot{V}_{O_2}$ measured from a baseline of zero load (see "Material and Methods") and the previously found value for the postural component of work during 1 limb exercise of 170 ml/min [9] the % figures for the arms versus the legs and 1 limb versus 2 limbs are reduced to 33% and 62% respectively. The changes in $\dot{V}_{E\,max}$ and $f_{H\,max}$, reflect these differences in $\dot{V}_{O_2\,max}$ and are also associated with the effective muscle (plus bone) mass used to perform single and 2-limb exercise (Table 1 and Fig. 2). The $\dot{V}_{E\,max}$ of the arms is half that of the legs whereas the $f_{H\,max}$ is reduced from 191 ± 5 beats per min to 166 ± 12 beats/min; a difference of 24 beats/min (Table 1). However, the actual maximal values of cardiac and ventilatory function recorded during 1 and 2 arm and 1 leg work in the present investigations also reflect the age of our subjects and the method used to measure $\dot{V}_{O_2\,max}$, particularly the time the subject was able to endure at maximal effort. An increase in $f_{H\,max}$ and $V_{E\,max}$ for example, in 1- or 2-arm work, could always be achieved by urging the subject to endure a further minute at or above a level of work beyond which the $\dot{V}_{O_2\,max}$ occurred. During work with the arms or 1 leg, all subjects gave local pain rather than circulatory or respiratory stress as a reason for their inability to continue work at maximal effort. Thus the large increase in the inter-subject variation of $f_{H\,max}$ as the effective muscle mass is reduced (Table 1) is probably more a reflection of the subjects ability to endure the 'local pain' and stress of maximal effort, than limits imposed by the circulation.

The criterion of maximal effort we have used as evidence of $\dot{V}_{O_2\,max}$ achievement also present certain difficulties. We found it impossible to measure the $\dot{V}_{O_2\,max}$ during a single session in 1- and 2-arm and 1-leg work and during preliminary experiments we could not discern a clear plateau of $\dot{V}_{O_2}$ with increasing work load which has hitherto been accepted as the major criterion of maximal effort in bicycle work with the legs. This necessitated developing a new criterion based on duplicate measurements of "$\dot{V}_{O_2\,max}$" agreeing within ± 5% when taken on separate occasions. This method of serial measurements of "$\dot{V}_{O_2\,max}$" undoubtedly suffers the disadvantage that the results may be influenced by repetition of

measurement, particularly if one fails to make satisfactory observations of $\dot{V}_{O_2\,max}$ on the first 2 occasions. The repeated maximal exercise may in itself provide a stimulus to cardiovascular conditioning and one may be changing or influencing the parameter which one is trying to measure. Secondly, in sedentary individuals who find the work stressful or fatiguing on the first occasion, it may prove difficult to persuade them to attend the laboratory on successive occasions. Thus ultimately one's measurements of $\dot{V}_{O_2\,max}$ in this type of exercise is biased towards the well motivated and co-operative subjects. In future studies this difficulty may be overcome by the development of secondary criteria (cf. Åstrand [2] for work with the legs).

During submaximal exercise it is clear that the f_H responses to arm and 1- and 2-leg work are distinct. For given $\dot{V}_{O_2}$ of 1.0 l · min^{-1} the f_H of arm work from a restrained position is approximately 40 beats/min higher than corresponding leg work.

At near maximal levels of work, the asymptotic nature of the $\dot{V}_{O_2}/f_H$ curve which characterises 2-leg exercise (see Fig. 4 and Davies [8]) is absent in arm and 1-leg work. This factor together with the intra-subject variability of $\dot{V}_{O_2}$ and f_H and the inter-subject variability of $f_{H\,max}$ already mentioned has a profound effect on accuracy with which the $\dot{V}_{O_2\,max}$ can be predicted in the different forms of exercise. During 2-leg work extrapolation of the $\dot{V}_{O_2}/f_H$ curve to a $f_{H\,max}$ of 195 beats/min underestimates the "true" or observed $\dot{V}_{O_2\,max}$ as previously shown [8] by 420 ± 180 ml · min^{-1} (Table 1). In 1-leg work, however, the situation is reversed, extrapolation of the $\dot{V}_{O_2}/f_H$ curve to a $f_{H\,max}$ of 175 beats/min overestimates observed $\dot{V}_{O_2\,max}$ by + 70 ± 200 ml · min^{-1}. The use of extrapolation procedures in 1- and 2-arm work also gives a realistic prediction of $\dot{V}_{O_2\,max}$, but the intersubject variability relative to absolute values of $\dot{V}_{O_2\,max}$ in these forms of exercise is increased (Table 1). The bias in estimation of $\dot{V}_{O_2\,max}$ for the different forms of work may be removed by using the appropriate regression equations relating predicted to observed $\dot{V}_{O_2\,max}$ (see Fig. 5 and Davies [8]), but the overall accuracy of predicting $\dot{V}_{O_2\,max}$ is still limited to approximately ± 8%, ± 15% and ± 23% for 1-leg, 2-arm and 1-arm work respectively (Table 1) compared to ± 5% for the directly observed values in the three forms of work (see "Material and Methods" and Fig. 1). If the observed $\dot{V}_{O_2\,max}$ of the arms is used to estimate the maximal performance with the legs, the order of accuracy is ± 12% [Eq. (iii)] which represents approximately a three-fold increase in the error given for the observed 1 to 2 leg $\dot{V}_{O_2\,max}$ conversion [Eq. (i)].

Thus, the results of this investigation suggest that a reliable and accurate estimation of work capacity may be made using single limb exercise provided direct observations of $\dot{V}_{O_2}$ at maximal performance

are carried out and the arms and legs are treated separately: 1 limb $\dot{V}_{O_2\,max}$ may be converted to an estimated 2 limb value with an accuracy of 4 to 7%. If the $\dot{V}_{O_2\,max}$ of 1-leg or arm is predicted from $\dot{V}_{O_2}/f_H$ measurements rather than measured directly, the resulting estimates give only a crude indication of the 'true' or observed performance. Finally, however, a word of caution: it must be appreciated that the measurements in this study were taken on subjects, who, though they were not in training in the conventional sense, nevertheless enjoy taking physical exercise. One feels sure that it would have been impossible to have made the required number of maximal observations on 12 subjects who had been selected at random from the community. Thus our values for 1 limb exercise are biased towards subjects with a sufficient interest in exercise and the motivation to see the investigation through to its conclusion. We do not know, as yet, how far our methods and results can be applied specifically to patients or may be taken as representative of the population at large. Further research will be required before these questions can be answered.

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Effects of Hypoxic Training on Normoxic Maximal Aerobic Power Output

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Abstract. The responses to 1-leg submaximal and maximal exercise have been studied in four male subjects before and after a 5 week training programme. One leg was trained under normoxic conditions and the other under hypoxic ($F_{IO_2} = 0.12$) conditions for 30 min/day, 3 times/week at a fixed absolute work load which approximated to 75 % of the limb's normoxic $\dot{V}_{O_2max}$. Before and after training both limbs were measured in normoxia, one limb was additionally measured in hypoxia. The aim of the experiments being to use each subject as his own control and to try and elucidate the effects of hypoxia per se as a training stimulus to the improvement of maximal aerobic power output ($\dot{V}_{O_2max}$) measured in normoxia.

The results showed that before training the responses to exercise at submaximal and maximal levels were identical in each limb; the effects of hypoxia being to raise $\dot{V}_{E\,1.5}$ and $f_{H\,1.5}$, to reduce $\dot{V}_{O_2max}$ and to leave $\dot{V}_{O_2\,450}$ unchanged. The effects of the two types of training were to reduce $\dot{V}_{O_2\,450}$, decrease $f_{H\,1.5}$ and increase $\dot{V}_{O_2max}$, the effects being independent of the F_{IO_2}. The changes in $\dot{V}_{O_2max}$ of the hypoxic and normoxic trained legs were related to the initial $\dot{V}_{O_2max}$ of each subjects' limb. It was concluded that our investigation lends no support to the view that hypoxia has either an additive or potentiating effect with exercise during a training programme on the improvement of aerobic power output measured under normoxic conditions.

Key words: Maximal Aerobic Power — Training — Hypoxia — 1-Leg Work.

Exposure to a reduced fractional concentration of oxygen in the inspired air over a prolonged period of time is known to give rise to an increased cardiac output at given oxygen intake, a rise in pulmonary ventilation, an enhanced plasma level of haemoglobin (and possibly myoglobin) concentration and a change in the degree of capillarisation in working muscle (see [1] for general review). All these factors are associated (under normoxic conditions) with a rise in maximal aerobic power output ($\dot{V}_{O_2max}$), but whether hypoxia itself, acts as a training stimulus is unknown. Some studies [15] have claimed to have shown an increase in sea level aerobic performance following prolonged exposure to and work at high altitude, but others [3] have not—the question remains open.

In the present study in order to overcome certain genetic and environmental (*e.g.* the initial starting level of fitness of different individuals [22]) factors which are known to confound the results of even the best planned training studies, we have studied four subjects during 1-leg exercise using each subject as his own control. One limb of each subject was trained under normoxic conditions and the other under hypoxic conditions ($F_{IO_2} = 0.12$) for 30 min/day; 3 times/week over a 5 week period at 75% of the limbs $\dot{V}_{O_2max}$ under normoxic conditions. Each limb was subjected to exactly the same absolute work intensity and since we have shown that a pair of limbs from a given subject are physiologically similar, the only variable factor in the training regimen was the degree of hypoxia. Thus any differences in the aerobic power output of a pair of limbs following training should be attributable to this factor alone and not masked by other biological variables.

Material and Methods

The material for this investigation were four healthy male subjects aged 24 to 36 years. Their physical characteristics were as follows: weight, 79.4 ± 11.6 kg; height, 180.2 ± 8.0 cm; lean body mass (estimated from skinfold thickness and body weight), 68.0 ± 7.6 kg; and limb muscle (plus bone) volume, 7.74 ± 0.67 l (right leg) and 7.34 ± 0.56 l (left leg). The anthropometric measurements were taken by methods previously described in detail [8]. The subjects were all interested in physical activity but none was in training before the commencement of the investigation. In preliminary experiments they were allowed to become accustomed to pedalling the bicycle with 1-leg and to breathing hypoxic gas mixtures. The details of the bicycle used for these experiments have been given elsewhere [8]. It was a fixed wheel ergometer and the subject's foot was placed in a plimsoll which was attached firmly to the pedal with bolts. No attempts were made to return the crank to the upright position with springs or by any other means (*c.f.* Freyschuss and Strandell [12]). The subjects were measured either in triplicate (SM and WP) or duplicate (AT and PI) during submaximal and maximal exercise before and after training. The criteria used as evidence for maximal aerobic power ($\dot{V}_{O_2max}$) having been reached has been outlined previously [6, 8, 9] and the same method was used in this study. The subject's legs were measured in normoxia and in addition the leg to be trained under hypoxic conditions was measured in hypoxia at the onset and end of the training programme. Thus a total of 60 measurements of 1-leg exercise performance was made on the four subjects during the period of the investigation. The subjects trained 3 times/week for 30 min at a set absolute work load (approximately 75 % of their normoxic 1-leg $\dot{V}_{O_2max}$ before training) for 5 weeks. One leg was trained under normal environmental conditions ($F_{IO_2} = 0.21$) and the other in hypoxia ($F_{IO_2} = 0.12$). Each training session was rigidly supervised and the work output and cardiac frequency recorded for each subject throughout the training sessions.

The physiological responses to exercise were measured in a conventional way by the open circuit technique using a mixing chamber to sample mixed expired gases [10]. Cardiac frequency was monitored using a differential amplifier coupled to a linear instantaneous rate meter [19]. The results for submaximal exercise were analysed and expressed in terms of minute ventilation ($\dot{V}_E$) at a $\dot{V}_{CO_2}$ of 1.5 l · min^{-1} ($\dot{V}_{E\,1.5}$), tidal volume (V_T) at $\dot{V}_E$ of 30 l · min^{-1} ($V_{T\,30}$), oxygen intake

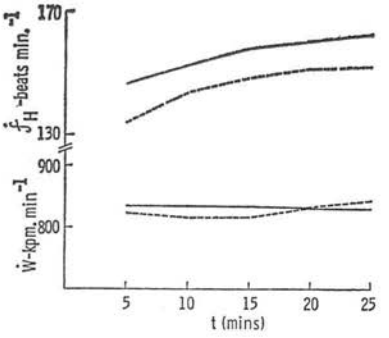

Fig. 1. The work output ($\dot{W}$ — kpm · min^{-1}) and cardiac frequency (f_H) of the leg trained in hypoxia (———) and normoxia (- - - - -). The lines represent the mean of 15 training sessions for each limb of the four subjects

($\dot{V}_{O_2}$) at a work output ($\dot{W}$) of 450 kpm · min^{-1} ($\dot{V}_{O_2\,450}$), cardiac frequency (f_H) at a $\dot{V}_{O_2}$ of 1.5 l · min^{-1} ($f_{H\,1.5}$) and a $\dot{V}_{O_2}$ at an f_H of 175 beats · min^{-1} [4, 8, 9]. During maximal exercise the mixing chamber was replaced by a Douglas bag and the subjects breathed directly into this via a low resistance (Otis-McKerrow) mouthpiece and a short piece (internal diameter $1^1/_2$") of smooth-bore tubing. The hypoxic mixture during the measurements before, during and after the training period was delivered to the subjects by rotameters (arranged in a way similar to that described by Cunningham *et al.* [5]) via a 500 l Douglas bag. The gas concentrations of the mixture was monitored continuously by automatic (paramagnetic O_2 and infra red CO_2) analysers.

Results

Throughout the period of the investigation the physical characteristics of the subjects showed no significant changes.

Submaximal Responses to Exercise

Before training the submaximal responses to training of each limb were identical under normoxic conditions (Table 1). The effects of hypoxia were to raise $\dot{V}_{E\,1.5}$ from 45.48 ± 4.85 to 57.59 ± 6.61 l · min^{-1} ($P < 0.001$) and $f_{H\,1.5}$ from 127 ± 11 to 146 ± 9 beats · min^{-1} ($P < 0.001$) but $\dot{V}_{O_2}$ for given $\dot{W}$ of 450 kpm · min^{-1} remained unchanged. In three subjects the increased $\dot{V}_{E\,1.5}$ was mainly due to an increase in V_T; f_H remaining unchanged, but in the remaining subject the opposite was true. Hypoxia reduced the $\dot{V}_{O_2}$ at an $f_{H\,175}$ from 2.51 ± 0.23 to 2.02 ± 0.18 l · min^{-1} ($P < 0.001$).

The effects of the two types of training were to reduce $\dot{V}_{O_2\,450}$ and $f_{H\,1.5}$, increase $\dot{V}_{O_2\,175}$ and leave $\dot{V}_{E\,1.5}$ and $V_{T\,30}$ unchanged. These changes appeared to be independent of whether the leg was trained in

Table 1. Submaximal training responses to 1-leg exercise. One leg of each subject was trained under normoxic conditions ("normoxic" leg), the other under hypoxic conditions ("hypoxic" leg). Both were measured in normoxia before and after training. In addition the "hypoxic" leg was measured in a similar way in hypoxia. The mean (± SD) data have been expressed in terms of a ventilation rate at a $\dot{V}_{CO_2}$ of 1.5 l · min^{-1} ($\dot{V}_{E\,1.5}$), tidal volume at a $\dot{V}_E$ of 30 l · min^{-1} ($V_{T\,30}$), a $\dot{V}_{O_2}$ at a work rate of 450 kpm · min^{-1} ($\dot{V}_{O_2\,450}$), cardiac frequency at a $\dot{V}_{O_2}$ of 1.5 l · min^{-1} ($f_{H\,1.5}$) and a $\dot{V}_{O_2}$ at an f_H of 175 beats/min^{-1} ($\dot{V}_{O_2\,175}$)

Variable	Units	Normoxia ($F_{IO_2} = 0.21$)				Hypoxia ($F_{IO_2} = 0.12$)	
		"normoxic" leg		"hypoxic" leg		"hypoxic" leg	
		before	after	before	after	before	after
$\dot{V}_{E\,1.5}$	l · min^{-1} BTPS	49.08 ±5.67	47.73 ±3.27	45.48 ±4.85	47.81 ±4.21	57.59**** ±6.61	61.40**** ±8.59
$V_{T\,30}$	l · BTPS	1.75 ±1.10	1.61 ±0.52	1.50 ±0.76	1.50 ±0.37	1.75 ±0.47	1.51 ±0.15
$\dot{V}_{O_2\,450}$	l · min^{-1} STPD	1.31 ±0.07	1.18*** ±0.07	1.31 ±0.06	1.14*** ±0.07	1.31 ±0.04	1.20 ±0.14
$f_{H\,1.5}$	beats · min^{-1}	131 ±10	118** ±10	127 ±11	118* ±13	146**** ±9	132**** ±8
$\dot{V}_{O_2\,175}$	l · min^{-1} STPD	2.36 ±0.21	2.86*** ±0.18	2.51 ±0.23	2.71* ±0.28	2.02**** ±0.18	2.44**** ±0.17

Significance: Before—after *** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$. Normoxia—hypoxia **** $P < 0.001$.

Table 2. Maximal responses to 1-leg exercise before and after training. Absolute ($\dot{V}_{O_2max}$) and net ($\dot{V}_{O_2max\ net}$) maximal aerobic power maximal ventilation ($\dot{V}_{E\,max}$) and cardiac frequency ($f_{H\,max}$). Mean (± SD) data as Table 1

Variable	Units	Normoxia ($F_{IO_2} = 0.21$)				Hypoxia ($F_{IO_2} = 0.12$)	
		normoxic leg		hypoxic leg		hypoxic leg	
		before	Δ after	before	Δ after	before	Δ after
$\dot{V}_{Emax}$	l · min^{-1} BTPS	129.0 ±2.4	+ 9.8 ±10.9	129.4 ±4.8	+11.3 ±11.4	129.3 ±8.8	— 2.45 ±20.90
$\dot{V}_{O_2max}$	l · min^{-1} STPD	2.83 ±0.12	+ 0.24** ± 0.20	2.79 ±0.13	+ 0.34** ± 0.19	2.25**** ±0.11	+ 0.21** ± 0.13
$\dot{V}_{O_2max\ net}$	l · min^{-1} STPD	2.34 ±0.12	+ 0.28** ± 0.16	2.34 ±0.09	+ 0.36** ± 0.19	1.87**** ±0.13	+ 0.18** ± 0.08
f_{Hmax}	beats · min^{-1}	177 ±7	+ 4 ± 5	177 ±8	+ 4 ± 7	179 ±7	— 1 ± 5

Significance: Before normoxic—hypoxic leg in normoxia, NS. Before normoxia—hypoxia ****$P < 0.001$.
Before and after training ** $P < 0.001$.

Analysis of variance before and after training in normoxia and hypoxia

	Df	SS	MS	F-ratio	Sign
Between subjects	3	0.0508	0.0169	2.32	NS
Between types of training	1	0.0149	0.0149	2.04	NS
Error	2	0.0146	0.0073		

hypoxia or normoxia (Table 1). Thus the relationship of the various submaximal physiological variables to each other for each limb remained unchanged before and after training.

Maximal Responses to Exercise

The changes in responses to maximal exercise before and after training are summarised in Table 2. Before training there was no significant differences between the right and left legs of the subjects. Following training the $\dot{V}_{E\,max}$, $\dot{V}_{O_2max\ net}$ and f_{Hmax} of the leg trained in normoxia changed by $+ 9.8 \pm 10.9\ l \cdot min^{-1}$; $+ 0.28 \pm 0.16$ ($P < 0.01$); and 4 ± 5 beats $\cdot$ min^{-1} respectively. The corresponding figures for the opposite limb trained in hypoxia were $+ 11.3 \pm 11.4\ l \cdot min^{-1}$; $0.36 \pm 0.19\ l \cdot min^{-1}$ ($P < 0.01$); and $+ 4 \pm 7$ beats $\cdot$ min^{-1}. An analysis of variance revealed no significant differences between the two limbs following the two different types of training.

The effects of hypoxic training on the aerobic power output of the leg which was measured before and after training in hypoxia (in addition to normoxia—see "Material and Methods") are also shown in Table 2. Before training, maximal exercise in hypoxia ($F_{IO_2} = 0.12$) resulted in $0.54\ l \cdot min$ (19%) decrease in 1-leg $\dot{V}_{O_2max}$.

Following training the changes in "hypoxic" $\dot{V}_{O_2max}$ both in absolute ($l \cdot min$) and relative (%) terms was less than the corresponding figures for the same "hypoxic trained" leg measured in normoxia. As a consequence of this the difference between the normoxic and hypoxic $\dot{V}_{O_2\,max}$ widens after training.

Discussion

Interest in hypoxia as a possible training stimulus was revived by the decision to hold the Olympic games at Mexico City in 1968 but since the initial spate of publications on the subject (see Margaria [18] for general review) discussion of the topic in the literature appears to have declined. Nevertheless, the major question remains: does exercise and hypoxia have an additive effect and thereby produce an improvement in performance over and above that produced by the same training in normoxia? If proven, the advantages to athletes are clear but an affirmative answer to the question may also have application to other fields, such as rehabilitation and occupational medicine, where one is often concerned with facilitating the patients' return to an active normal life, within a short a time-span as possible.

Klausen *et al.* [17] were the first to show that altitude induced hypoxia may have a substantial affect on $\dot{V}_{O_2max}$ on return to sea level. They observed in 12 ♂ subjects a 14% increase in $\dot{V}_{O_2max}$ after a 5 week stay

at altitude of 3800 m. Their results found agreement in the later work of Dill and Adams [11] and the study of Bannister *et al.* [2] who used an environmental chamber to simulate altitude conditions. In contrast, Hansen *et al.* [16] and Buskirk *et al.* [3] found no change in $\dot{V}_{O_2max}$ on return to sea level following training and exposure to altitude. However, all these studies are difficult to interpret for various reasons. The investigations of Klausen, Buskirk, Dill and Bannister used no controls, whereas the study of Hansen took this precaution but failed to standardise the training regimen adequately at altitude and sea level so that the effects of training were not directly comparable. The only study, to our knowledge, which used control subjects and made attempts to gain equivalence between the hypoxic training at altitude and normoxic training at sea level is that of Roskamm *et al.* [20]. They related the training programme to the initial fitness of their subjects and measured three groups of ♂s under simulated conditions at altitudes of 3450 m, and 2250 m and at 260 m. Their data indicate mean changes of + 10.0% and + 17.5% in $\dot{V}_{O_2max}$ measured at 260 m for those who trained at the simulated altitudes compared with 6.4% for those who were trained and measured at 260 m.

We have approached the problem in an entirely different manner. Using our previous experience in 1-leg work [8] we designed our experiments to see if we could assess the influence of hypoxia per se on maximal aerobic power output. The use of 1-leg exercise enabled us to use each subject as his own control whilst training under both normoxic and hypoxic conditions. The training stimulus in terms of absolute work output performed was constant for both legs, throughout the training programme and the $\dot{V}_{O_2max}$ of each subjects' right and left leg before training commenced was closely similar (Table 2). Thus the two most important criteria for assessing the effects of a training programme were met: a constant training stimulus under controlled environmental (F_{IO_2}) conditions and the standardisation of the confounding influence of the initial level of fitness of a subject on his subsequent improvement in $\dot{V}_{O_2max}$ performance [22]. Under these circumstances it is clear that the effect of hypoxia per se on aerobic power improvement is minimal. We find no clear evidence that training in reduced F_{IO_2} enhances or potentiates the improvement of $\dot{V}_{O_2max}$ as observed under normoxic conditions (Table 2).

Hypoxia per se, as has been shown many times [1], increases $f_{H\,1.5}$ and $\dot{V}_{E\,1.5}$ for given $\dot{V}_{O_2}$ and reduces $\dot{V}_{O_2max}$ but has little or no effect on the O_2 cost of work. Training in hypoxia though it has the effect of changing the former three parameters in the opposite direction in the "hypoxic" leg (see Table 1) when the magnitude of the changes are compared to normoxia, the differences between f_H and $\dot{V}_E$ in the two conditions

remain unchanged whilst the *difference* in $\dot{V}_{O_2max}$ actually shows a small (4%) but significant increase after training (Table 2; *c.f.* Saltin [21]). The reduction of $\dot{V}_{O_2max}$ in hypoxia as found by Gleser [14] is associated with the fall in F_{IO_2} (Table 2). This is in direct contrast to the findings for the effect of hyperoxia on 1 limb performance [8].

The effects of hypoxia per se on $\dot{V}_{O_2max}$ must be borne in mind when interpreting our observations on the respective merits of training under hypoxic and normoxic condition. We chose to require each subject to exercise at a constant absolute work level which represented approximately 75% of his normoxic $\dot{V}_{O_2max}$. Thus under hypoxic conditions the relative work performed rose to nearer 90% during the greater part of the training period. Since the relative (as well as the absolute) work intensity is known to have an effect on the improvement of $\dot{V}_{O_2max}$ observed in a training programme [7] a critic of our results may argue that our data are biased in favour of the limb trained in hypoxia. However, though we would accept such criticism as fair, we would point out that the additional benefits of training at intensities of exercise beyond 80% $\dot{V}_{O_2max}$ has not been documented and is open to question [1] and in any case such arguments would only strengthen our case. Since despite the possibility of a training bias towards hypoxia analysis of co-variance of our data clearly reveals no statistical evidence for a difference in the two forms of training stimuli.

This is an important finding but one must be careful not to extend beyond the experimental conditions under which it has been established. Firstly, it must be emphasised that our data were obtained from exercise with 1 limb. We have argued [8] previously that the essential limitation to such exercise is probably peripheral and not central and more dependent upon flow to, and utilization of oxygen within, the leg's muscle rather than the output of the heart. It therefore seems unlikely that our results can be applied directly to other forms of work and in particular, normal 2-legged cycling or running. Secondly, the fact that we have shown no differences to exist between "hypoxic" and "normoxic" training cannot be interpreted to mean that exercise in hypoxia is necessarily without effect. Our experiments were of relatively short duration (5 weeks) and Fusancho *et al.* [13] have clearly shown the importance of duration of exposure to hypoxia and age at which it is initiated on the changes observed at altitude. One cannot say what the effects might have been if our experiments had been prolonged, or younger (or older) subjects had been used. Certainly it would be difficult to explain the high values of $\dot{V}_{O_2max}$ found in high altitude natives [23] without invoking some of these factors. Thus, we cannot refute unequivocally the theory that training in hypoxia or at altitude has a beneficial effect on sea level physical performance but our present results seriously question

its physiological basis. Our experiments indicate that provided the absolute work stimulus is constant, the training effect of cycling is independent of the F_{IO_2} in which the limb is trained.

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Changes in physiological performance of the lower limb after fracture and subsequent rehabilitation

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Summary

1. Eight patients who had suffered a fracture of one leg were studied before and after a 7 weeks period of rehabilitation during work with one leg and both legs on a bicycle ergometer.

2. In submaximal exercise minute ventilation for a given carbon dioxide output and tidal volume at a given minute ventilation remained unchanged throughout the period of therapy for both one- and two-leg exercise: oxygen intake for a given work output and cardiac frequency for a given oxygen intake decreased in both the injured and uninjured limb during one-leg work, although in two-leg exercise there was no significant change.

3. Oxygen intake at zero load was subtracted from the maximum oxygen intake measured during loaded exercise to give net values for each limb exercised separately or both legs exercised together. The net maximum oxygen intake thus calculated increased 8·9% (+0·17 l/min) in the uninjured leg and 17·4% (+0·29 l/min) in the injured leg during one-leg exercise. In two-leg exercise the increase was 17·2% (+0·43 l/min), which approximately equals the increase in the two legs measured separately.

4. In both legs there was an increase in leg muscle (plus bone) volume although this was significant in the injured leg only.

5. The maximum oxygen intake attained in two-leg exercise for a given leg volume in the patients at discharge was not significantly different from that found previously in a cross-sectional survey of young healthy (naval) servicemen. Thus the rehabilitation programme investigated appears to be effective, although the spontaneous recovery without a rehabilitation programme is unknown.

Key words: aerobic power, rehabilitation, fracture, exercise, muscle.

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Introduction

In a previous paper (Davies & Sargeant, 1975a) we described the effects of fracture and subsequent immobilization of the leg on the exercise tolerance of twenty-five patients on admission to the Joint Services Medical Rehabilitation Unit at Chessington, Surrey. The present study was designed to follow the changes in physiological performance of the lower limbs of eight of these patients, weekly during an intensive course of rehabilitation therapy. The course included exercises specifically (but empirically) designed to improve the mobility, strength and aerobic capacity of the patients. All the patients were servicemen, the therapy lasted on average 7 weeks and was supervised by trained remedial gymnasts.

Material and methods

All eight patients had suffered fractures of one leg only and these were divided as follows: five fractures of the tibia and fibula (three right and two left); one fracture of the left femur, and two cases where femur and tibia and fibula fractures were sustained (both left legs). The average period spent immobilized was 105 days and exercise tests were first given 25

TABLE 1. *Physical characteristics of the eight patients before and after a 7 weeks' course of rehabilitation*

Age, height, weight, sum of four skinfold thicknesses (Σsf), lean body mass (LBM) and leg volume (LV) of the injured and uninjured limb are given as mean values ±SD. Significance, before and after therapy: $^{**}P<0.01$.

	Age (years)	Height (cm)	Weight (kg)	Σsf (mm)	LBM (kg)	LV (l)	
						Uninjured	Injured
Before	21·8 ±3·7	172·9 ±5·8	70·2 ±13·3	35·5 ±11·3	59·21 ±9·38	6·38 ±0·80	5·66 ±0·94
After	21·9 ±3·7	172·8 ±6·0	71·2 ±14·6	33·6 ±13·6	60·48 ±9·42	6·65 ±1·08	6·23** ±1·04

days after the end of immobilization, which was on average 7 days after the commencement of rehabilitation therapy at Chessington. Their physical characteristics before and after the period of rehabilitation are given in Table 1.

The methods used have been described in detail elsewhere (Davies & Sargeant, 1975a), and therefore only a brief outline of the procedure will be given here. The patients were studied at 7–10 day intervals. One- and two-leg exercises were performed on a 'fixed wheel' stationary bicycle ergometer.

Measurements were made on each occasion of the responses to a progressive exercise test of each leg separately and of two legs combined with a rest of at least 1 h between each test, the aim in each test being to span the subject's capacity from zero load up to and, where possible, including maximum in four or five work loads.

At submaximal work levels, oxygen intake ($\dot{V}O_2$), carbon dioxide output ($\dot{V}CO_2$), minute ventilation ($\dot{V}_E$), and cardiac (f_H) and respiratory (f_R) frequencies were measured over the last 2 min of each 5 min work load by a standard open-circuit technique (Davies & Sargeant, 1974). Owing to the difficulty of obtaining maximal measurements in one-leg exercise (particularly in patients) which meet the criteria developed for two-leg exercise, it was sometimes necessary to take duplicate measurements on subsequent days and to assess the results in the light of secondary criteria based on our previous observation of a maximum cardiac frequency of ~175 beats/min in one-leg work. The maximal aerobic power output ($\dot{V}O_{2\ max.}$) was expressed in absolute terms (l/min) as directly measured and as a net value ($\dot{V}O_{2\ max.\ net}$) calculated from the $\dot{V}O_{2\ max.}$ minus the $\dot{V}O_2$ of pedalling at constant speed against zero load (see Davies & Sargeant, 1974 for further details). In addition, height and weighs were measured on each visit to the laboratory at well as four skinfold thicknesses (suprailiac, subscapular, triceps and biceps) for the estimation of lean body mass (Durnin & Rahaman, 1967). Leg (muscle plus bone) volume (LV) measurements by physical anthropometry were also made after the technique developed by Jones & Pearson (1969) (see Davies & Sargeant, 1975b, for further details and validation of the technique for use with patients undergoing rehabilitation therapy).

To overcome difficulties of habituation to and learning (Davies, Tuxworth & Young, 1970) of the standard one- and two-leg exercise tests, all the patients were required to perform the work test before definitive measurements were taken.

Results

The changes in physical characteristics and responses to submaximal exercise are summarized in Tables 1 and 2.

During the period of rehabilitation, body weight, height and lean body mass (LBM) remained unchanged (Table 1). The major effect of therapy was to increase the mean (±SD) leg muscle (plus bone) volume (LV) of the uninjured (6·38 ±0·80 litres to 6·65 ±1·08 litres) and injured (5·66 ±0·94 litres to 6·23 ±1·04 litres; $P<0.01$) legs, though at the end of rehabilitation there was still a significant difference ($P<0.01$) between the two limbs.

In response to submaximal exercise (Table 2) minute ventilation at $\dot{V}CO_2$ 1·5 l/min ($\dot{V}_{E\ 1.5}$) and tidal volume at $\dot{V}_E$ 30 l/min ($V_{T\ 30}$) during exercise with the injured, uninjured and two legs combined were similar at the beginning of therapy and were not significantly different from the values found

TABLE 2. *Changes in submaximal responses to exercise after rehabilitation*

Results shown are: minute ventilation at $\dot{V}CO_2$ 1·5 l/min ($\dot{V}_{E\,1\cdot5}$); tidal volume at $\dot{V}_E$ of 30 l/min ($V_{T\,30}$); oxygen intake at a work load of 73 W in one-leg work ($\dot{V}O_{2\,(73)}$), or 147 W in two-leg work ($\dot{V}O_{2\,(147)}$); cardiac frequency at $\dot{V}O_2$ 1·5 l/min ($f_{H\,1\cdot5}$); oxygen intake at f_H 175 beats/min in one-leg ($\dot{V}O_{2\,(175)}$) and 195 beats/min in two-leg exercise ($\dot{V}O_{2\,(195)}$). Significance: $*P<0\cdot05$; $***P<0\cdot001$.

Variable	Units	One-leg exercise		Two-leg exercise
		Uninjured	Injured	
$\dot{V}_{E\,1\cdot5}$	l/min	−3·20	+2·29	+2·23
$\dot{V}_{T\,30}$	l	−0·03	+0·32	+0·05
$\dot{V}O_{2\,(73/147)}$	l/min	−0·01	−0·06	−0·04
$f_{H\,1\cdot5}$	beats/min	−12*	−18***	−6
$\dot{V}O_{2\,(175/195)}$	l/min	+0·49***	+0·49***	+0·16

previously on a larger group of servicemen studied on single occasions at Chessington (Davies & Sargeant, 1975a). Rehabilitation had no effect on the ventilatory responses to work; indeed the relationship of $\dot{V}_E$ to $\dot{V}CO_2$ and V_T to $\dot{V}_E$ before and after therapy for the different forms of exercise could be adequately represented by equations (1) and (2).

$$\dot{V}_E\,(l/min_{BTPS}) = -0\cdot45 + 32\cdot69\ \dot{V}CO_2\ (l/min_{STPD}) \quad (r = 0\cdot97;\ SD = 6\cdot93\ l/min) \qquad (1)$$

$$\dot{V}_E\,(l/min_{BTPS}) = -20\cdot95 + 46\cdot56\ V_T\ (l_{BTPS}) \quad (r = 0\cdot89;\ SD = 11\cdot20\ l/min) \qquad (2)$$

At the onset of therapy $\dot{V}O_2$ for given work output ($\dot{W}$) of 73 W was higher in exercise with the injured (1·50 ± 0·12 l/min) than with the uninjured leg (1·37 ± 0·09 l/min; $P<0\cdot001$). The effect of rehabilitation was to reduce the oxygen cost of work in exercise with either leg producing a small rise in mechanical efficiency (Fig. 1)

The mechanical efficiency of two-leg work was unaffected by rehabilitation and throughout the investigation was closely in accord with previous results found for normal healthy subjects in this laboratory. The cardiac frequency values showed similar changes to those described for $\dot{V}O_2$. At the beginning of the rehabilitation period cardiac frequency at $\dot{V}O_2$ 1·5 l/min ($f_{H\,1\cdot5}$) was 150 ± 21 beats/min in exercise with the injured leg compared with 139 ± 18 beats/min ($P<0\cdot001$) with the uninjured leg. At the end of therapy these values in exercise with the uninjured and injured legs were reduced to 127 ± 9 beats/min and 132 ± 14 beats/min respectively, but the difference between them was still significant ($P<0\cdot05$). The predicted $\dot{V}O_2$ at f_H 175 beats/min changes from 2·22 ± 0·44 l/min to 2·71 ± 0·35 l/min in exercise with the uninjured leg

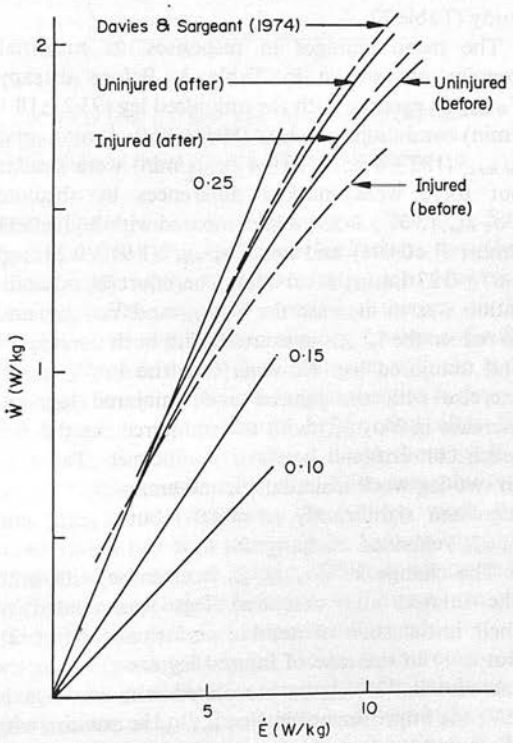

FIG. 1. Relationship of work output ($\dot{W}$) to aerobic energy expenditure (E) expressed in terms of W/kg body weight for the injured and uninjured limbs of patients before and after rehabilitation. Isopleths for mechanical efficiency and previous one-leg exercise data (Davies & Sargeant, 1974) from normal healthy subjects are also shown.

TABLE 3. *Changes in response to maximal exercise after rehabilitation*
Pulmonary minute ventilation ($\dot{V}_{E\ max.}$), absolute ($\dot{V}O_{2\ max.}$) and net ($\dot{V}O_{2\ max.\ net}$) maximum oxygen intake and cardiac frequency ($f_{H\ max.}$) values are shown as mean values. Significance: ***$P<0.001$; **$P<0.01$; *$P<0.05$.

Variable	Units	One-leg exercise		Two-leg exercise
		Uninjured	Injured	
$\dot{V}_{E\ max.}$	l/min	+9·7	+7·7	+8·8
$\dot{V}O_{2\ max.}$	l/min	+0·17	+0·33***	+0·42**
$\dot{V}O_{2\ max.\ net}$	l/min	+0·17	+0·29***	+0·43***
$f_{H\ max.}$	beats/min	−7*	−6**	+3

and 1·97 ± 0·43 l/min to 2·46 ± 0·38 l/min with the injured leg over the period of therapy. Both these changes were highly significant ($P<0.001$), as were the differences in the values in exercise with the injured compared with the uninjured leg both before ($P<0.001$) and after ($P>0.05$) rehabilitation. In contrast, in two-leg work the $f_{H\ 1.5}$ and $\dot{V}O_{2\ (195)}$ remained almost constant over the period of the study (Table 2).

The mean changes in responses to maximal exercise are shown in Table 3. Before therapy $\dot{V}_{E\ max.}$ in exercise with the uninjured leg (93·2 ± 18·1 l/min) and injured leg (89·9 ± 17·0 l/min) and $f_{H\ max.}$ (182 ± 6 and 183 ± 4 beats/min) were similar but there were marked differences in absolute $\dot{V}O_{2\ max.}$ (2·35 ± 0·30 l/min compared with 2·11 ± 0·34 l/min; $P<0.001$) and net $\dot{V}O_{2\ max.}$ (1·91 ± 0·27 and 1·67 ± 0·27 l/min; $P<0.001$). The effect of rehabilitation was to increase the $\dot{V}_{E\ max.}$ and $\dot{V}O_{2\ max.}$ and to reduce the $f_{H\ max.}$ in exercise with both the injured and uninjured leg. However, the rise in $\dot{V}_{E\ max.}$ in exercise with the injured and uninjured leg and increase in $\dot{V}O_{2\ max.}$ with the uninjured leg did not reach conventional levels of significance (Table 3). In two-leg work a similar picture emerged; $\dot{V}O_{2\ max.}$ increased significantly ($P<0.001$) but $\dot{V}_{E\ max.}$ and $f_{H\ max.}$ remained unchanged.

The change in $\dot{V}O_{2\ max.\ net}$ in exercise with both the injured and uninjured legs was related to their initial state of aerobic performance (Fig. 2), but only in the case of injured leg exercise was the increase in $\dot{V}O_{2\ max\ net}$ associated with increase in LV; the improvement in $\dot{V}O_{2\ max.\ net}$ in exercise with the uninjured limb appeared to be largely independent of LV (Fig. 3). The relationship of $\dot{V}O_{2\ max.}$ during one-leg (injured and uninjured) exercise to that during two-leg exercise after rehabilitation was described by the linear regression equation (3).

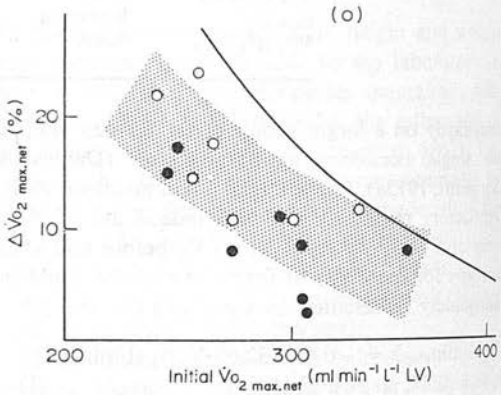

FIG. 2. Relationship of change in $VO_{2max.}$ ($\Delta\dot{V}O_{2\ max.}$%) to initial $\dot{V}O_{2\ max.}$ in ml min^{-1} l^{-1} (L$\dot{V}$). ——, Data for young healthy male subjects taken from Davies & Sargeant (1975c). The stippled area represents the limits (mean ±SD) of the curve for patients in the present study. ○, Injured leg; ●, uninjured leg. One injured leg value (○), which appeared to fall outside the normal range for these patients is excluded.

$$\text{Two-leg } \dot{V}O_{2\ max.}\ (\text{l/min}) = 0{\cdot}191 + 1{\cdot}23\ \text{one-leg } \dot{V}O_{2\ max.}\ (\text{l/min}) \quad (r = 0{\cdot}90;\ \text{SD} = 0{\cdot}22\ \text{l/min}) \tag{3}$$

This equation did not differ significantly from that found previously (Davies & Sargeant, 1974) for normal healthy adults. The relationship of two-leg $\dot{V}O_{2\ max.}$ to LV is shown in Fig. 4.

Discussion

The results before therapy commenced are in agreement with data previously published by us on a larger anthropometric and exercise cross-sectional study of new entrants to the Rehabilitation Unit at Chessington (Davies & Sargeant, 1975a, b) and therefore require no further discussion. We have also alluded to the main difficulties of measuring maximal aerobic power in patients recovering from

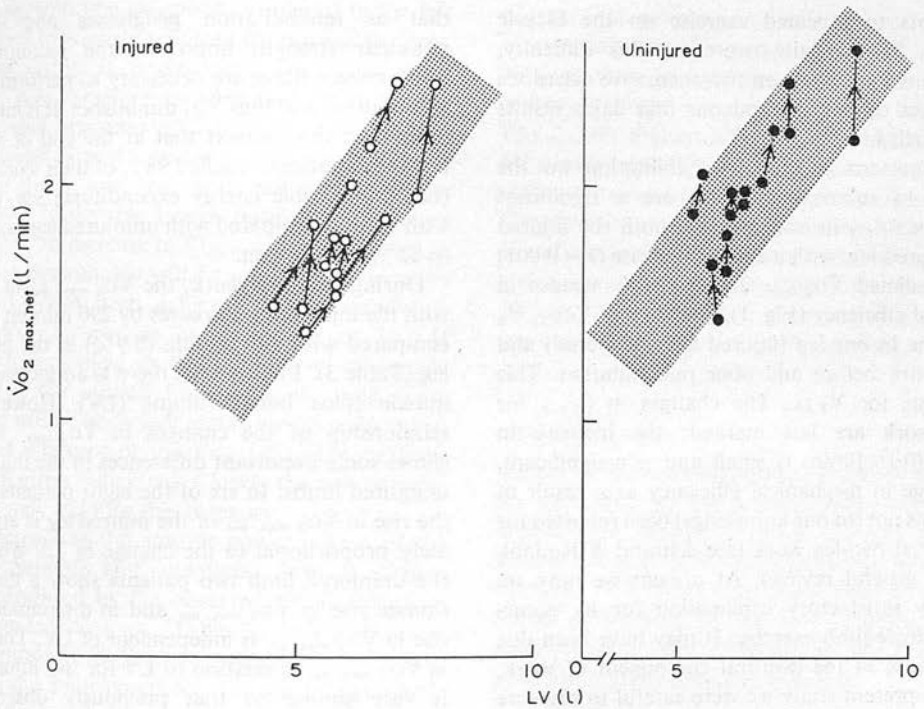

FIG. 3. Relationship of net maximal aerobic power ($\dot{V}O_{2\ max.}$) of the injured (○) limb and the uninjured (●) limb before and after rehabilitation to estimates of leg muscle (plus bone) volume (LV). The arrows indicate the direction of change. The shaded area represents the 95% confidence limits for the relationship previously found in normal subjects (Davies & Sargeant, 1974).

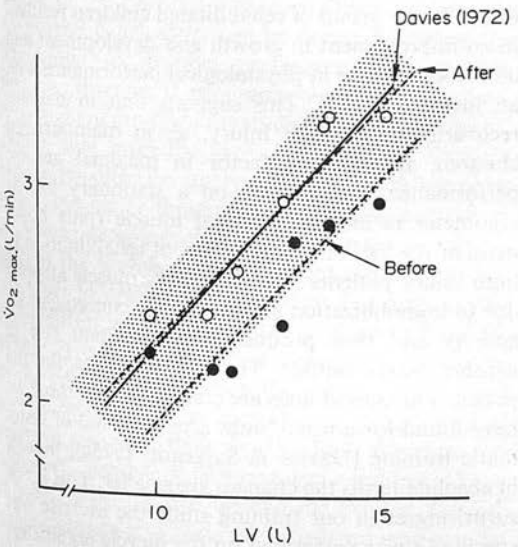

FIG. 4. Relationship of net $\dot{V}O_{2\ max.}$ during two-leg exercise before (●) and after (○) rehabilitation. ——, Regression line for service personnel data previously collected (Davies, 1972). The shaded area represents the 95% confidence limits for normal data (Davies, Mbelwa, Crockford & Weiner, 1973).

limb injuries in our previous paper (Davies & Sargeant, 1975a) and the same limitations described there apply to the present investigation. However, in studying the effects of a period of therapy on the responses to standard exercise one further difficulty is encountered, that of familiarization with the task. In many investigations where the exercise responses of a patient are measured before and after the administration of physical (or drug) therapy, it is often tacitly assumed that the differences observed are due to the treatment. In two-leg work this assumption has been shown to be open to question (Davies, Tuxworth & Young, 1970); on exposure to repeated tests, patients habituate to the given exercise and their physiological responses change.

In the present study we found it impractical to subject the patients to a comprehensive habituation programme as both the patients and their instructors were anxious to commence (or to continue if the subjects were weight-bearing) the programme of rehabilitation. The instructors thought (rightly) that they could not afford a break of 2 weeks in the already condensed programme whilst we subjected

the patients to repeated exercise on the bicycle ergometer. To partially overcome this difficulty, observations were made on two successive occasions at the onset of therapy and our first day's results were discarded.

The important effects of rehabilitation on the responses to submaximal work are a significant decrease in $f_{H\ 1.5}$ in exercise with both the injured and uninjured leg, with a consequent rise ($P < 0.001$) in the predicted $\dot{V}O_{2\ max.}$ and a small increase in mechanical efficiency (Fig. 1). For a given $\dot{V}CO_2$, $\dot{V}_E$ is the same in one-leg (injured and uninjured) and two-leg work before and after rehabilitation. This is also true for $V_{T\ 30}$. The changes in $f_{H\ 1.5}$ for two-leg work are less marked; the increase in $\dot{V}O_{2\ (195)}$ (0·16 l/min) is small and non-significant.

A change in mechanical efficiency as a result of training has not (to our knowledge) been reported for conventional two-leg work (see Åstrand & Rodahl, 1970, for general review). At present we have no completely satisfactory explanation for its occurrence in single-limb exercise. It may have been due to a decrease in the postural component of work, but in the present study we were careful to measure changes of mechanical efficiency from a baseline of zero load (see the Material and Methods section). The postural component of one-limb work has previously been found (Davies & Sargeant, 1974) to be a factor only at the highest levels of work and it will be noted (Fig. 1) that the shape of the relationship between work output and aerobic energy expenditure does not change before and after rehabilitation. The only marked decline in $\dot{V}O_2$ for given $\dot{W}$ we have observed previously is for subjects performing repeated negative (eccentric) work on a motor-driven treadmill (Davies & Barnes, 1972). In this study we postulated that the effect may be due to the decrease in the number of active muscle fibres required to perform the work (see Abbott, Bigland & Ritchie, 1952). It may be that a similar effect is occurring in one-leg work. To pedal a fixed-wheel cycle ergometer with one limb requires a high degree of muscular strength, particularly in the quadriceps, in order to return the pedal to upright position with each revolution. Normal subjects as well as patients often complain of local (rather than general cardiovascular) fatigue and muscular soreness as limiting factors to one-limb exercise. Undoubtedly the lower limbs of patients recovering from injury are relatively weak (see e.g. Zohn, Leach & Stryker, 1964; Cuddingham, 1973) and it is entirely conceivable that as rehabilitation progresses and dynamic muscular strength improves, the recruitment of fewer muscle fibres are necessary to perform a given work output and thus $\dot{V}O_2$ diminishes. It is interesting to note in this context that at the end of rehabilitation the patients reached 98% of their work output (but not aerobic energy expenditure; see Table 3) with injured compared with uninjured leg as opposed to 85% at the onset.

During maximal work, the $\dot{V}O_{2\ max.\ net}$ in exercise with the injured leg increases by 290 ml/min (17·4%) compared with 170 ml/min (8·9%) in the uninjured leg (Table 3). In both legs there is an increase in leg muscle (plus bone) volume (LV). However, the relationship of the changes in $\dot{V}O_{2\ max.}$ with LV shows some important differences in the injured and uninjured limbs. In six of the eight patients (Fig. 3) the rise in $\dot{V}O_{2\ max.\ net}$ of the injured leg is approximately proportional to the change in LV whereas in the uninjured limb two patients show a disproportionate rise in $\dot{V}O_{2\ max.\ net}$ and in the remainder the rise in $\dot{V}O_{2\ max.\ net}$ is independent of LV. The change in $\dot{V}O_{2\ max.\ net}$ in relation to LV for the injured limb is very similar to that previously observed for malnourished children (Davies, 1974). In this latter study the $\dot{V}O_{2\ max.}$ during cycling decreased *pari passu* with the loss of leg muscle (plus bone) volume. Further it was possible to show that improved diet in a different group of rehabilitated children resulted in an improvement in growth and development and an associated rise in physiological performance with an increase in LV. This suggests that in patients recovering from limb injury, as in malnourished children, the limiting factor in maximal aerobic performance during work on a stationary bicycle ergometer is the reduction of muscle (plus bone) mass of the leg. The major effect of rehabilitation in limb injury patients is to reverse the muscle atrophy due to immobilization plaster and the consequent inactivity and thus produce a concomitant rise in aerobic power output. The maximal data for the patient's uninjured limb are consistent with those we have found for normal limbs after a period of systematic training (Davies & Sargeant, 1975c), though in absolute terms the changes are smaller. This is not surprising as in our training study the exercise was specific (it was performed on the bicycle ergometer) and of high intensity and controlled daily under laboratory conditions. However, it is noteworthy that, with these patients, although the changes in $\dot{V}O_{2\ max.\ net}$ when related to their initial $\dot{V}O_{2\ max.\ net}$

values are, with one exception, displaced to the left of the curve previously reported for normal subjects, the relationship is nevertheless of similar form (Fig. 2). It is therefore possible that there may be two essential processes governing the physiological improvement of performance in patients recovering from leg injury and that both are related to the initial level of aerobic power output of the lower limbs: first, an increase in $\dot{V}O_{2\ max.}$ due to change in LV, which presumably will be self-limiting once the original LV has been achieved, and, secondly, a rise in $\dot{V}O_{2\ max.}$ *per se* independently of LV. If therapy is prolonged or the initial level of muscle atrophy in the injured limb is small, both these processes will be observed, giving rise to a change in slope and intercept of the $\dot{V}O_{2\ max.\ net}$/LV relationship for a given individual. Clearly the two patients in Fig. 3 may fall into this category.

The increase in the aerobic power output of the patients' injured and uninjured legs is almost exactly paralleled by the observed rise in the combined (two-leg) $\dot{V}O_{2\ max.}$ (Table 3). This again is in direct contrast to findings on normal subjects where training (and improving) the $\dot{V}O_{2\ max.}$ of each limb individually had minimal effect on the $\dot{V}O_{2\ max.}$ of the two limbs combined during work on the bicycle ergometer (Davies & Sargeant, 1975c). However, for reasons mentioned earlier regarding the specific nature of the previous one-leg training used by us, no general conclusions can be drawn regarding the limitations to aerobic performance in patients receiving therapy. It may seem reasonable to argue, in the light of our previous study (Davies & Sargeant, 1975c), that since the increase in two-leg $\dot{V}O_{2\ max.}$ exactly parallels the improvement of each leg combined, the major limitation to maximal exercise in patients undergoing rehabilitation must be peripheral (within the muscle tissue) and not central (cardiovascular) in origin. The argument is supported by the relatively small change in $f_{H\ 1.5}$ for two-leg work (Table 2) but it does ignore one important point: in the present investigation the patients were given specific exercises during their therapy programme to improve not only their injured limb function but also their general cardiovascular condition. Thus it is not surprising that we find the two-leg $\dot{V}O_{2\ max.}$ value increases *pari passu* with the combined one-leg $\dot{V}O_{2\ max.}$.

Finally, from a practical point of view the rehabilitation course as presented at the Joint Services Medical Rehabilitation Unit, achieves its stated object of returning servicemen to normal duty. At the end of rehabilitation the patients are able to achieve 95% of their aerobic power output with injured compared with uninjured leg and the two-leg $\dot{V}O_{2\ max.}$ for a given LV (Fig. 4) is not significantly different from that found in a previous cross-sectional survey of normal (naval) servicemen (Davies, 1972). The spontaneous recovery occurring without benefit of a rehabilitation programme is, however, unknown and is an area needing further investigation if the efficacy of rehabilitation programmes such as the one studied is to be accurately assessed.

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We are indebted to the patients and their instructors for their cheerful and willing co-operation in this investigation and to Group Captain E. Ward, Group Captain C. B. Wyn-Parry and Wing Commander C. D. Evans for their support and provision of facilities at the Joint Services Medical Rehabilitation Unit, Chessington. Mr S. M. Riggs gave invaluable technical assistance.

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Effects of training on the physiological responses to one- and two-leg work

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DAVIES, C. T. M., AND A. J. SARGEANT. *Effects of training on the physiological responses to one- and two-leg work.* J. Appl. Physiol. 38(3): 377–381. 1975.—The effects of training resulting from one-leg exercise on a stationary bicycle ergometer have been studied. Seven subjects were habituated to one- and two-leg progressive exercise tests on 11 successive days and were then trained for 60 min·day^{-1} (30 min each leg) 3 times per wk for 5–6 wk at ~80% of their one-leg $\dot{V}O_{2\,max}$. $\dot{V}E_{max}$ increased ($P < 0.05$) by ~14 l·min^{-1} and $\dot{V}O_{2\,max}$ by ~0.34 l·min^{-1} (+14%; $P < 0.05$) in one-leg exercise. This latter increase was not, however, reflected in the two-leg $\dot{V}O_{2\,max}$ which only increased 145 ml·min^{-1} (4.7%). It was concluded that training is specific and in one-leg work the phenomenon is mainly peripheral in origin, but in two-leg work the limitation to maximal exercise is still provided by the capacity of the central cardiovascular system to transport oxygen to a given effective muscle mass.

exercise; $\dot{V}O_{2\,max}$; single-limb work; leg muscle (plus bone) volume; repeated exercise

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FROM THE RESULTS of previous work by Davies, Tuxworth, and Young (7) it was suggested that the initial changes in the physiological responses to repeated exercise may be due to readjustments in central circulatory control and the redistribution of cardiac output in favor of the working muscles. Our data suggested that the effects of repeated work could be divided into two stages; the first four occasions in which there was a marked change in the cardiovascular responses to exercise without a concomitant change in maximal aerobic power output ($\dot{V}O_{2\,max}$) and the second where a gradual change in $\dot{V}O_{2\,max}$ and a slow decline in cardiac frequency for given oxygen intake were observed. We felt that these two processes (though possibly interrelated) were sufficiently distinct to warrant the terms habituation and training.

In the present experiments we have attempted to examine more specifically the nature of the training stimulus together with the possible role of central and peripheral cardiovascular factors underlying the phenomenon. To investigate these questions we have used our experience with one-limb work (4, 5) and have studied the sequential effects of repeated exercise, first with the right leg, then with the left, and finally with both legs combined. To our knowledge only Gleser (11) has studied the effects of training in one-leg work prior to this investigation. He studied six sedentary subjects (mean $\dot{V}O_{2\,max}$ = 46.6, range 37.9 – 51.5 ml·kg^{-1}·min^{-1}), twice weekly, training at 75% $\dot{V}O_{2\,max}$ over a period of 4 wk. His results demonstrated large changes in aerobic power output but they are difficult to interpret on two points. *1*) The subjects performed the one-leg work in pairs and shared the work load by standing each side of the bicycle and pedaling with the inside leg and using the other leg for support. The observed physiological responses to exercise will obviously depend on the degree of cooperation between subjects though the author claims to overcome this difficulty through practice and familiarization with the involved procedure. Nevertheless, *2*) except for one subject (*EW*), his data suggest that the more sedentary subjects improved least and a plot of his $\Delta\dot{V}O_{2\,max}$ against initial $\dot{V}O_{2\,max}$ values reveals a curvilinear relationship which is completely the opposite to that found for normal two-leg work (see Saltin et al. (18)).

METHODS

The subjects were first habituated to exercise. They performed submaximal exercise on the first four occasions with one-leg (left or right), the second four occasions with the other leg, and finally the last three occasions with both legs. Following the period of habituation definitive submaximal and maximal one- and two-leg exercise measurements were taken immediately prior to, and at the cessation of, the training period. During one-leg exercise the foot of the active limb was fitted with a plimsoll which was secured to the bicycle pedal by bolts and two metal plates. The standard Monark stationary bicycle ergometer had a fixed wheel and no attempt was made to return the "passive" pedal during cycling. In both one- and two-leg exercise care was taken to ensure that saddle height was correctly adjusted on the first occasion of measurement and maintained throughout the period of the study. At submaximal work loads oxygen intake ($\dot{V}O_2$), minute ventilation ($\dot{V}E$), carbon dioxide output ($\dot{V}CO_2$), and respiratory (f_R) and cardiac frequencies (f_H) were measured during the last 2 min of a 5-min period by the standard open-circuit techniques (6) using a dry gas (Parkinson Cowan), paramagnetic (Servomex), and infrared (Hilger Watts) meters for ventilation volumes and O_2 and CO_2 concentrations, respectively. Maximum determinations were made and assessed using criteria previously developed (2, 4, 5) by the standard Douglas bag technique, a collection being taken over the last minute of a final 3-min work load during which the subjects were encouraged to pedal as hard as possible. If they were able to

sustain this load the level was raised again and a second collection taken.

Mechanical efficiency (defined as the ratio of work performed to aerobic energy expenditure) was calculated from the $\dot{V}O_2$ work data using a base line of pedaling against zero load (13, 20).

The training of each subject was supervised in the laboratory. They trained for 60 min·day^{-1} (30 min with each leg), 3 times per wk over a 5- to 6-wk period at ~80% of their (one-leg) $\dot{V}O_{2\,max}$. During each training period work output and cardiac frequency were monitored continuously.

During the study the subjects were measured for height and weight, and the sum of four skinfold thicknesses taken for the estimation of lean body mass after the method of Durnin and Rahaman (8). In addition leg volume (LV, muscle plus bone) was measured according to the method of Jones and Pearson (14). The physical characteristics are summarized in Table 1.

RESULTS

During the period of habituation to exercise, minute ventilation at a carbon dioxide output of 1.5 l·min^{-1} ($\dot{V}E_{1.5}$), tidal volume at a minute ventilation of 30 l·min^{-1} (VT_{30}) and oxygen intake at a work level of 450 kpm/min ($\dot{V}O_{2\,450}$) remained essentially constant. At the commencement of measurement these parameters averaged 50.38 ± 6.84 l·min^{-1}, 1.14 ± 0.05 l, and 1.43 ± 0.12 l·min^{-1} with the first leg and 49.23 ± 2.20 l·min^{-1}, 1.09 ± 0.17 l, and 1.44 ± 0.11 l·min^{-1} with the second leg, respectively. At the conclusion of the study these parameters had changed by −6.09 and −2.96 l·min^{-1}, −0.08 and +0.10 l, and −0.03 and −0.02 l·min^{-1} for the first and second legs, respectively. On the other hand, $f_{H\,1.5}$ remained constant during the first two occasions with first leg and then showed small but significant ($P < 0.05$) (−5 beats·min^{-1}) fall on the third occasion and thereafter remained constant. The small change in $f_{H\,1.5}$ (−6 beats·min^{-1}) was observed in the second leg but following the first (and not second) occasion. However, the magnitude of the mean changes in f_H was partly masked by the fact that two of the subjects were athletic and the $\Delta f_{H\,1.5}$ observed were related to the initial level of $\dot{V}O_{2\,max}$ of the subject. The changes in $f_{H\,1.5}$ were more marked in the two sedentary subjects, the results from one of these is shown in Fig. 1. During two-leg work (occasions 9–11 inclusive), all the physiological responses to exercise measured remained constant. The responses to maximal one-leg work measured after 1st, 4th, and 8th occasions and two-leg work after 9th and 11th occasions, showed no significant changes: $\dot{V}E_{max}$, $\dot{V}O_{2\,max\,net}$ and $f_{H\,max}$ for one- and two-leg work remained essentially constant throughout the period of habituation.

The effects of 5- to 6-wk training program on the maximal responses of each leg is shown in Table 2. Before and after training there were no significant differences between the right and left leg of the subjects. The major effect of training was to effect an increase in $\dot{V}E_{max}$ (right leg) from 123.41 ± 10.60 to 140.73 ± 15.33 l·min^{-1} and (left leg) from 125.32 ± 14.93 to 136.50 ± 10.74 l·min^{-1} ($P < 0.05$), and the $\dot{V}O_{2\,max\,net}$ (right leg) from 2.43 ± 0.41 to 2.82 ± 0.43 l·min^{-1} ($P < 0.05$) and (left leg) 2.43 ± 0.42 to 2.72 ± 0.39 l·min^{-1} ($P < 0.05$); but $f_{H\,max}$ re-

TABLE 1. *Physical characteristics*

	Age, yr	Ht, cm	Wt, kg	Σ4sf, mm	LBM, kg	LV, l	
						RL	LL
Before training (n = 7)	29.1 ±5.5	176.3 ±8.1	74.9 ±10.4	30.1 ±7.7	64.8 ±7.7	7.33 ±0.79	7.11 ±0.58
Δ After	0.1 ±0.7	0.1 ±1.34	0.11 ±3.81	−0.03 ±1.46	−0.22 ±0.61	+0.20 ±0.29	−0.06

Values are means ± SD. Σ4sf = sum of 4 skinfold thicknesses (biceps, triceps, subscapular, suprailiac); LBM = lean body mass; LV = leg muscle (plus bone) volume; RL and LL = right and left leg.

TABLE 2. *Effects of training on maximal one-leg minute ventilation rate, net aerobic power output, and cardiac frequency in seven subjects*

	Before			After		
	$\dot{V}E_{max}$, l·min^{-1}	$\dot{V}O_{2\,max\,net}$, l·min^{-1}	$f_{H\,max}$, beats·min^{-1}	$\dot{V}E_{max}$ l·min^{-1}	$\dot{V}O_{2\,max\,net}$, l·min^{-1}	$f_{H\,max}$, beats·min^{-1}
Right leg	123.41 ±10.60	2.43 ±0.44	178 ±7	140.73 ±15.33	2.82* ±0.43	179 ±9
Left leg	125.32 ±14.93	2.43 ±0.42	178 ±6	136.50† ±10.74	2.72* ±0.39	180 ±9
Two legs	152.39 ±17.32	3.06 ±0.52	190 ±7	148.6 ±6.36	3.20 ±0.44	187 ±8

Values are means ± SD. * Significance (before-after): $P < 0.01$. † $P < 0.05$.

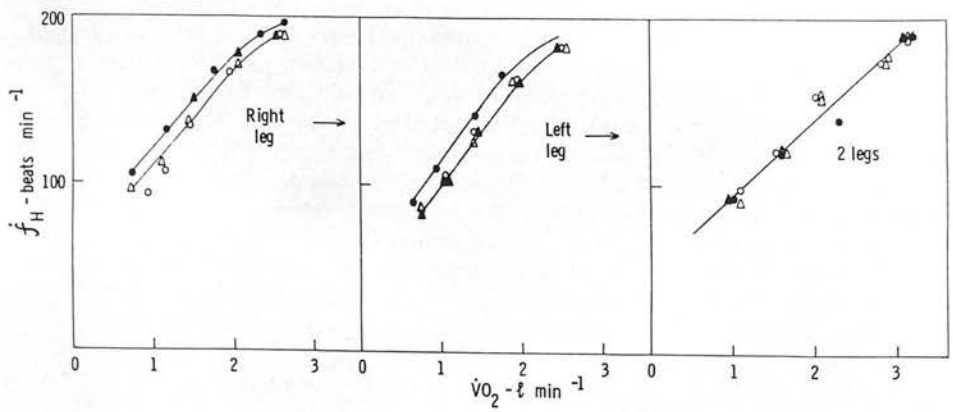

FIG. 1. Cardiac frequency (f_H) in relationship to $\dot{V}O_2$ (*subj SR*) during period of habituation. Measurements were made sequentially: *a*) right leg, *b*) left leg, and *c*) two legs combined. *Day 1* (●), *day 2* (▲), *day 3* (○), and *day 4* (△).

mained unchanged at approximately 179 beats·min^{-1} throughout the period of the investigation.

The mean change of ~340 ml·min^{-1} in $\dot{V}O_{2\ max\ net}$ represents an improvement in aerobic performance of 14% for the group of subjects as a whole, but individually the % $\Delta\dot{V}O_{2\ max\ net}$ is clearly related to the initial level of $\dot{V}O_{2\ max\ net}$ expressed in ml·l(LV)$^{-1}$·min^{-1} (Fig. 2). The relationship is curvilinear ($P < 0.05$) and is adequately represented by the equation

$$\dot{V}O_{2\ max\ net}\ (\%) = 17.48 - 0.813\ (\text{initial}\ \dot{V}O_{2\ max\ net}\ \text{ml·l(LV)}^{-1}\text{·min}^{-1}) + 0.00097\ (\text{initial}\ \dot{V}O_{2\ max\ net}\ \text{ml·l(LV)}^{-1}\text{·min}^{-1})^2$$

SD = 5.3%

The increase in $\dot{V}O_{2\ max\ net}$ with training is not accompanied by a concomitant increase in leg muscle (plus bone) volume. For a given rise in $\dot{V}O_{2\ max\ net}$, LV remains almost constant (Table 1), thus the linear regression line relating the two variables shows a parallel displacement to the left of the 'normal' relationship (Fig. 3).

In contrast to the large changes in $\dot{V}O_{2\ max\ net}$, of one-leg, the improvement in two-leg $\dot{V}O_{2\ max\ net}$ (following the training of each limb individually) was relatively small (Table 2). If the improvement of each $\dot{V}O_{2\ max\ net}$ limb was additive one would have expected a change in two-leg $\dot{V}O_{2\ max\ net}$ of the order of 671 ml·min^{-1} (22%), in fact, the measured change was only 145 ± 242 ml·min^{-1} (4.7%). This lack of improvement of two-leg $\dot{V}O_{2\ max\ net}$ following one-leg training has a profound effect on the relationship between the two variables. Before training, one-leg $\dot{V}O_{2\ max}$ was 79% of two-leg $\dot{V}O_{2\ max}$ and the linear regression relationship lay within the limits previously reported (4) for normal healthy subjects. Following training the $\dot{V}O_{2\ max\ net}$ of one leg rose to 87% of two-leg $\dot{V}O_{2\ max}$ and the line relating the two variables was displaced to the right. The respective equations relating one- and two-leg $\dot{V}O_{2\ max\ net}$ were before training

$$\text{two-leg}\ \dot{V}O_{2\ max\ net}\ (\text{l·min}^{-1}) = 0.05 + 1.229\ \text{one-leg}\ \dot{V}O_{2\ max\ net}\ (\text{l·min}^{-1})$$

after training

$$\text{two-leg}\ \dot{V}O_{2\ max\ net}\ (\text{l·min}^{-1}) = 0.32 + 1.042\ \text{one-leg}\ \dot{V}O_{2\ max\ net}\ (\text{l·min}^{-1})$$

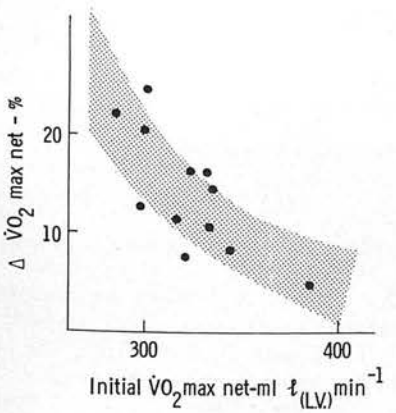

FIG. 2. Relationship of change in $\dot{V}O_{2\ max}$ (Δ%) to initial level of $\dot{V}O_{2\ max}$ for one-leg work, initial $\dot{V}O_{2\ max}$ has been expressed in terms of ml·l(LV)$^{-1}$·min^{-1}.

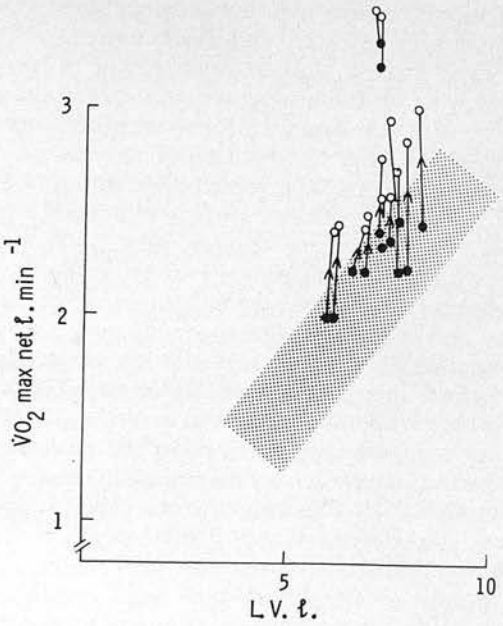

FIG. 3. Relationship of $\dot{V}O_{2\ max\ net}$ to estimates of limb muscle (plus bone) volume (LV) before (●) and after (○) training. Shaded area represents previous limits (mean ± SD) for normal subjects (4).

DISCUSSION

The present experiments were designed to evaluate the effects of training during work on a stationary bicycle ergometer.

Our data for habituation of one limb differ in some important respects from that previously reported (7) for two-limb work. In the present investigation, the $\dot{V}O_2$ for given $\dot{W}$, the V_T and $\dot{V}E$ for given $\dot{V}CO_2$ (as before for two-limb work) remain constant, but there is a much smaller change in $f_{H\ 1.5}$ and the sequence of events differs from those previously recorded. In two-leg work (7) there was a marked fall in cardiac frequency of 21 beats·min^{-1} from occasion 1 to 4; half the change being observed following the first exposure of the subjects to exercise. By comparison in the present experiments the change in $f_{H\ 1.5}$ was not seen until the second occasion of measurement and was reduced in magnitude to an average of 5 beats·min^{-1} for the four subjects. This small mean change in $f_{H\ 1.5}$ was not transferred directly to the second limb, but the prior habituation of one limb appeared to facilitate that of the other: as the same order of change in $f_{H\ 1.5}$ appeared following the first occasion of measurement of the second leg (Fig. 1). During the period of habituation the one-leg $\dot{V}O_{2\ max}$ remained unchanged. When the subjects performed two-leg work having habituated each limb individually and sequentially, there was no evidence of change in the physiological responses to repeated submaximal work, $\dot{V}O_{2\ 900}$, $\dot{V}E_{1.5}$, V_{T30} and $f_{H\ 1.5}$ remained constant. These findings cannot necessarily be extrapolated to other forms of exercise and markedly different types of subjects. In this context it should be noted that the present group of subjects were physically active and accustomed to performing laboratory experiments although they had not previously pedaled a bicycle ergometer: this contrasts with and may explain

some of the differences with the previous study (7) where the subjects were sedentary and totally naive.

The large mean (±14%) improvement of $\dot{V}O_{2\,max}$ of 1 leg following an intensive 5-wk period of training is in agreement with that found by Gleser (11), but our results differ decisively from his in that our changes are closely associated with the initial aerobic power output of the limb (Fig. 2). The $\dot{V}O_{2\,max}$/initial $\dot{V}O_{2\,max}$ relationship parallels that found previously for two-leg work in young (aged 20–30 yr) male (18) and older (+40 yr) subjects (9, 19). The training effect appears to be specific to the leg being exercised and is not transferred to the contralateral limb (cf. Klausen et al. (16), for arm and leg work). However, the combined improvement (22%) of each limb is not reflected by a concomitant increase in $\dot{V}O_{2\,max}$ of both legs when they are used together to pedal the ergometer. Following the training of each leg the change in two-leg $\dot{V}O_{2\,max}$ is minimal (~5%). This supports our view (4) and those of others (see Bevegård and Shepherd (1) for review) that the limiting factor to exercise where relatively large muscle groups are employed, such as in two-leg cycling, is the ability of the cardiovascular system to transport the required volume and not the capacity of the muscles to utilize oxygen.

The present results (Table 2) cannot be reconciled with those (e.g., 15) who argue that the increase in $\dot{V}O_{2\,max}$ during rhythmic two-leg exercise is solely limited by peripheral (tissue) events within the working muscle and unrelated to cardiovascular transport and the capacity of the heart to increase its output. Certainly we agree with Gleser (11) that in exercise where the effective muscle mass is reduced (as in one-leg work) and presumably $\dot{Q}$ is not limiting the case for peripheral improvement of $\dot{V}O_{2\,max}$ as found in the present experiments is overwhelming, but on the basis of one-leg–two-leg improvement we fundamentally disagree with him, that this can be used as argument for the unique peripheral nature of the training stimulus, in fact, quite the reverse.

Clearly in any integrated system such as the (cardiovascular) transport and (muscle) utilization of oxygen, it is unwise to argue purely in terms of peripheral and central events and this is not our case. Our view is solely that the improvement in $\dot{V}O_{2\,max}$ will depend ultimately on the balance between tissue and cardiovascular events. Where the effective muscle mass is limiting the balance will swing towards the periphery, but in work demanding larger muscle groups, it will move toward the delivery of oxygen from the central circulatory system and the limits imposed by $\dot{Q}$. It would seem axiomatic that transport and utilisation are the two fundamental links in the chain of improvement of maximal aerobic power, but the degree to which they are integrated will depend on the type of exercise and the state of training of the subject.

The displacement of the $\dot{V}O_{2\,max\,net}$(one-leg):LV relationship following training again illustrates very clearly the point we have made several times (see Davies (3) for review) regarding the essential independence of leg muscle (plus) bone volume and aerobic power output. The $\dot{V}O_{2\,max\,net}$ of the limb improves whilst the LV remains almost constant (Table 1 and Fig. 3). The large change in $\dot{V}O_{2\,max\,net}$ following training demonstrates the great capacity of leg muscle to improve its oxidative capacity. In Fig. 3 we have also included the data on one extremely fit athlete (a marathon runner) and it will be seen that even his initial value $\dot{V}O_{2\,max\,net}$ before training lies well outside the normal limits for trained (but not athletic) men. Following training the improvement in $\dot{V}O_{2\,max}$ was small (<4%) which is to be expected (Fig. 2), but it is intriguing to speculate how an athlete can reach a $\dot{V}O_{2\,max}$ of over 3 $l \cdot min^{-1}$ with one limb, which has a LV of just over 7 l. Theoretical consideration, for example, of limb blood flow would seem to suggest a rate in excess of previously observed maximal two-leg work values. Clearly if our results from one subject can be confirmed a study of the haemodynamic responses of high (aerobic) performance athletes during single leg exercise would well repay further study.

Thus, in summary our results show that one limb can be trained independently of the other and of the volume of muscle (plus bone) present but the improvement in aerobic performance is not reflected in the two limbs when they are combined. This gives rise to the labile nature of the one- to two-leg $\dot{V}O_{2\,max}$ relationship already noted (p. 8 and Davies and Sargeant (4)), and further underlies the specificity of the training process. Further research is required to elucidate the initial effects of repeated exercise but taking our combined findings for two-leg (7) and one-leg work (present investigation) together it may well be that habituation is merely the earliest manifestation of a more general training phenomenon. Unfortunately this would suggest that the first time (and on each successive occasion) you subject a hitherto sedentary subject to exercise in the laboratory you change his physiological state. This may well confound the very variables you are trying to measure.

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Physiological Responses to Exercise in Patients Following Fracture of the Lower Limb

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PHYSIOLOGICAL RESPONSES TO EXERCISE IN PATIENTS FOLLOWING FRACTURE OF THE LOWER LIMB

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ABSTRACT. **Twenty-five patients with healed fractures of the lower limb and nine normal control subjects were measured anthropometrically and during maximal and submaximal one- and two-leg bicycle exercise. Oxygen intake at a given submaximal work level of 450 kmp min^{-1} and cardiac frequency at an oxygen intake of 1.5 l min^{-1} were significantly higher ($p<0.001$) in the injured compared with the uninjured limbs of the patients and normal subjects. The maximum aerobic power of the injured and uninjured limbs of the patients were 18.8 % (0.44 l min^{-1}) and 25.6 % (0.61 l min^{-1}) respectively lower than the right and left legs of the control subjects. The corresponding value for 2-leg work was 17.6 % (0.51 l min^{-1}). The deterioration in 1-leg performance of the patients was associated with a concomitant decrease in leg muscle (plus bone) volume. In 2-leg work this factor was also probably combined with a deterioration in performance due to general cardiovascular deconditioning.**

INTRODUCTION

In previous work from this laboratory we have shown that during cranking the stationary bicycle ergometer with the arms (4) or with one- (5) or two-leg(s), (3) a close association exists between maximal exercise performance and the effective muscle (plus bone) volume used to perform the work. The data were collected on normal healthy subjects but to our knowledge no systematic studies of this type have been applied to patients undergoing rehabilitation from limb injury.

Following a bone fracture, there is loss of muscular power and atrophy of the affected limb. This loss of function may be partially due to hospitalization of the patient and the immobilization of the limb in post-operative plaster. Courses of rehabilitation are designed to reverse these changes and aim to return the patient as soon as possible to a normally active life. However, few scientific data are available on these points, and indeed, little is known of the combined effects of limb fracture and immobilization on the subsequent performance and aerobic power output of the muscle.

In the present paper we have examined 25 patients with healed fracture(s) of the lower limb, 50 days following removal of plaster together with a small group of 9 healthy normal male subjects. Submaximal (and in some cases maximal) one-leg and two-leg exercise were performed; the uninjured limb measurements of each patient were used for control purposes.

MATERIAL

The patients were 25 male servicemen with leg fracture: they were studied at the beginning of a full time course of rehabilitation at the Joint Services Medical Rehabilitation Unit, Chessington. All patients had suffered fractures of one leg only and these were divided as follows: fractures of femur, 2 left and 3 right; fractures of tibia (and fibula), 6 left and 12 right; and finally in two cases, (both left legs) combined fractures of the femur and tibia. Of these 25 cases, 10 had some form of internal fixation. The causes of fracture were sport (10 cases), motor accidents (6), accidental falls (5), parachuting accidents (3) and gunshot wound (1). The average period spent immobilized was 105 days (range 35–205) and patients were seen when they first entered Chessington or as soon as they were fully weight bearing and had sufficient joint mobility to pedal a bicycle without discomfort or pain. Thus the patients were seen on average 50 days after the end of immobilization and 18 days after commencing therapy at Chessington.

For comparison data on 4 of the patients' instructors and 5 laboratory assistants were collected. The instructors and laboratory workers were on average 7 years older than the patients, their physical details together with those of the patients are included in Table I.

METHODS

During the first visit to the laboratory, subjects were measured for height and weight. Skinfold measurements were

Table I. *Physical characteristics of the 25 patients, 4 normal instructors and 5 laboratory assistants*

Weight (wt); height (ht); sum of 4 skinfold thicknesses (Σ sf.); lean body mass (LBM) calculated from Σ sf. after the method of Durnin & Rahaman (9); limb (muscle plus bone) volume (LV—see Jones & Pearson (12) and Davies & Sargeant (6) of the uninjured (u) and injured (i) legs.

Subjects	Age (yr)	Wt (kg)	Ht (cm)	Σ sf. (mm)	LBM (kg)	LV (litres) u	LV (litres) i
Patients (n=25)	22*** ±4	69.8 ±11.4	174.2 ±6.5	31.8 ±9.2	59.7 ±8.7	6.49+++*** ±0.92	5.76*** ±0.86
Normals (n=9)	29 ±6	75.3 ±9.2	176.0 ±7.4	29.6 ±6.6	65.2 ±6.9	7.38 ±0.79	7.29 ±0.70

Significance: patients cf. normals ***$P<0.001$; patiens L.V. (u cf. i) +++$P<0.001$.

taken at four sites according to the method of Durnin & Rahaman (9) to enable lean body mass to be estimated. The volume of both legs (muscle plus bone) was measured after the anthropometric technique developed by Jones & Pearson (12). The validity of this technique as applied to limbs which have undergone muscle atrophy due to immobilization is discussed in detail elsewhere (6).

In order to reduce the effects of learning and habituation to the task (8) the subjects were allowed to exercise on the first visit to the laboratory; the definitive measurements were taken during the second visit.

During the definitive measurements both patients and normal subjects were required to exercise in three sessions with a rest period of not less than 1 hour between each pedalling a standard Monark stationary bicycle ergometer with each leg separately and then with 2 legs. During 1-leg exercise the foot of the active limb was fitted with a plimsoll which was secured to the bicycle pedal by bolts and two metal plates. The bicycle had a fixed wheel and no attempt was made to return the "passive" pedal during cycling (10). Care was taken to ensure that the saddle height was correctly adjusted to enable the pedalling to be performed with maximal extension of the leg. The subjects were required to pedal the ergometer in a progressive test starting from zero load and increasing up to and sometimes including maximal levels. The submaximal work loads were chosen to give 4 or sometimes 5 work levels. In submaximal experiments, oxygen intake ($\dot{V}O_2$), minute ventilation ($\dot{V}_E$) and respiratory (f_R) and cardiac frequencies (f_H) were measured during the last two minutes of a 5 minute period by the standard open circuit technique (1) using dry gas (Parkinson Cowan Ltd.) paramagnetic (Servomex Ltd.) and infra red (Hilger Watts Ltd.) meters for ventilation volume and O_2 and CO_2 analysis respectively. Mechanical efficiency (defined as the ratio of work performed, $\dot{W}$, to aerobic energy expenditure) was calculated from the $\dot{V}O_2$ $\dot{W}$ data using a baseline of pedalling against zero load (11, 14).

Where the subjects were able to exercise at maximum, the final collection was taken using the standard Douglas Bag technique over the last minute of a 3 minute work load during which the subject was encouraged to pedal at maximal effort. If this could be sustained the load was raised again and a second collection taken.

Criteria for maximal performance

In the normal control subjects performing 2-leg exercise the "$\dot{V}O_2$ plateau" criterion for the attainment of maximum aerobic power ($\dot{V}O_{2\ max}$) was applied (1). However, in two-leg work with the patients and with one leg work in both patients and controls, we found as previously (5) that this criterion was not easy to apply. The patients in particular although they were "encouraged" to keep going as long as possible, found it possible to sustain maximal or near maximal effort for relatively short periods. In order, in cases of doubt, to overcome this difficulty, we took duplicate measurements of $\dot{V}O_2$ on subsequent days and assessed the results in conjunction with secondary criteria based on our previous observation of a maximum cardiac frequency of 175 beats/min in one leg work (5). If the patient complained of pain either at the fracture site or in the joints the exercise was discontinued and the results not included.

RESULTS

The responses to submaximal exercise are shown in Table II.

For a given $\dot{V}CO_2$, $\dot{V}_E$ is identical for exercise with the injured and uninjured limb of patients and does not differ significantly from that of normal subjects. The $\dot{V}O_2$ at a $\dot{W}$ of 450 kpm min^{-1} is significantly higher ($P<0.001$) in the injured limb compared with the uninjured limb of the patients and the normal limbs of the instructors and laboratory workers. The raised $\dot{V}O_2$ for a given $\dot{W}$ in the injured limb is also associated with a decline in the predicted $\dot{V}O_2$ at an f_H of 175 beats min^{-1}. The $\dot{V}_{T\ 1.5}$ is similar for both limbs of the patients but significantly lower ($P<0.001$) than that observed for the normal subjects.

In 2-leg exercise the V_T and $\dot{V}O_{2\ 195}$ are again significantly higher ($P<0.001$) in the normals and $f_{H\ 1.5}$ lower but $\dot{V}O_{2\ 900}$ and $\dot{V}_{E\ 1.5}$ are similar in both groups.

Table II. *Submaximal responses to 1- and 2-leg work*

The data have been expressed in terms of pulmonary minute ventilation ($\dot{V}_E$) at a fixed carbon dioxide output of 1.5 l min^{-1} ($\dot{V}_{E\,1.5}$);tidal volume at a $\dot{V}_E$ of 30 l min^{-1} ($V_{T\,30}$); oxygen intake ($\dot{V}O_2$) at a work load of 450 kpm min^{-1} ($\dot{V}O_{2\,450}$) for 1-leg and 900 kpm min for 2-leg work ($\dot{V}O_{2\,900}$), cardiac frequency at a $\dot{V}O_2$ of 1.51 min^{-1} ($f_{H\,1.5}$) and a $\dot{V}O_2$ at an f_H of 175 beats min ($\dot{V}O_{2\,175}$ 1-leg) and 195 beats min ($\dot{V}O_{2\,195}$ 2-legs)

(*a*) 1-*leg*	$\dot{V}_{E\,1.5}$ l min^{-1} B.T.P.S.	$V_{T\,30}$ l B.T.P.S.	$\dot{V}O_{2\,450}$ l min^{-1} S.T.P.D.	$f_{H\,1.5}$ beats min^{-1}	$\dot{V}O_{2\,175}$ l min^{-1} S.T.P.D.
Patients (n=25)					
Uninjured leg	50.46 ±9.00	1.14 ±0.18	1.40*** ±0.11	142*** ±15	2.16** ±0.37
Injured leg	50.54 ±7.23	1.12 ±0.15	1.51 ±0.11	153 ±19	1.98 ±0.42
Normals (n=9)					
Right leg	46.87 ±4.07	1.52+++ ±0.94	1.35 ±0.08	123++ ±13	2.66++ ±0.39
Left leg	46.94 ±3.81	1.85+++ ±0.96	1.36+++ ±0.07	124+++ ±9	2.58+++ ±0.32
(*b*) 2-*legs*	$\dot{V}_{E\,1.5}$ l min^{-1} B.T.P.S.	$V_{T\,30}$ l. B.T.P.S.	$\dot{V}O_{2\,900}$ l min^{-1} S.T.P.D.	$f_{H\,1.5}$ beats min^{-1}	$\dot{V}O_{2\,195}$ l min^{-1} S.T.P.D.
Patients (n=13)	44.56 ±4.78	1.21 ±0.23	2.13 ±0.15	140 ±18	2.74 ±0.62
Normals (n=9)	42.63 ±7.15	2.34+++ ±1.07	2.12 ±0.11	121++ ±12	3.55 ±0.40

Significance: Patients uninjured cf. injured leg: $^{**}P<0.01$; $^{***}P<0.001$.
Patients cf. normals (1-leg and 2-leg): $++P<0.01$; $+++P<0.001$.

At maximal exercise (Table III) the $\dot{V}_{E\,max}$ and $\dot{V}O_{2\,max}$ of the patients for both 1-leg and 2-leg exercise are significantly ($P<0.001$) lower than their normal counterparts but the $f_{H\,max}$ if comparable in the two groups. In the normal subjects there is no difference between the $\dot{V}O_{2\,max}$ of the right and left leg but in the patients the net aerobic power of the injured limb is 10.6% lower then the uninjured limb. Comparison of the limbs of the patients with those of the normal subjects shows larger differences: 18.8% (0.44 l min^{-1}) uninjured leg and 25.6% (0.61 l min^{-1}) injured leg. For 2-leg work the reduction of $\dot{V}O_{2\,max}$ in the patients was 17.6% (0.51 l min^{-1}).

The association of net $\dot{V}O_{2\,max}$ with estimate of leg (muscle plus bone) volume is shown in Fig. 1. The reduction in $\dot{V}O_{2\,max\,net}$ of the injured leg of patients is associated with a decline in LV. For a given LV the $\dot{V}O_{2\,max\,net}$ of the uninjured and injured legs of patients and the right and left leg of the control subjects lie within previously found normal limits for young healthy subjects. Though the data for the patients lie below and on average are displaced to the right of their normal counterparts. Thus inpatients, the decline in 1-leg $\dot{V}O_{2\,max\,net}$ is partly a function of the change in leg muscle (plus bone) volume.

The relationship of 1-leg to 2-leg $\dot{V}O_{2\,max}$ of the patients and normal subjects is shown in Fig. 2. It can be seen that whilst the uninjured leg to 2-leg relationship in the patients is similar, though somewhat lower in absolute values, to the normal subjects the injured leg to 2-leg relationship is significantly displaced to the right ($P<0.05$).

DISCUSSION

The submaximal and maximal exercise observations on the patients and normal subjects in this study were made under standardised laboratory conditions.

The normal subjects were 5 laboratory workers and 4 remedial gymnasts. They were somewhat older, heavier, taller, less fat and had larger LV's, than the patients (Table I). However, the LV data for the left and right legs of the normal subjects were not significantly different from those previously pub-

Table III. *Responses to maximal exercise*

Maximal ventilation ($\dot{V}_{E\,max}$) absolute ($\dot{V}O_{2\,max}$) and net ($\dot{V}O_{2\,max\,net}$—see methods), oxygen intake and cardiac frequency ($f_{H\,max}$)

	Patients ($n=15$)				Normals ($n=9$)				
	$\dot{V}_{E\,max}$ l min^{-1} B.T.P.S.	$\dot{V}O_{2\,max}$ l min^{-1} S.T.P.D.	$\dot{V}O_{2\,max\,net}$ l min^{-1} S.T.P.D.	$f_{H\,max}$ beats min^{-1}	$\dot{V}_{E\,max}$ l min B.T.P.S.	$\dot{V}O_{2\,max}$ l min^{-1} S.T.P.D.	$\dot{V}O_{2\,max\,net}$ l min^{-1} S.T.P.D.	$f_{H\,max}$ beats min^{-1}	
Uninjured leg	97.6 ±16.0	2.35*** ±0.29	1.89*** ±0.24	179 ±6	125.7+++ ±10.6	2.81+++ ±0.17	2.33+++ ±0.17	174 ±5	Right leg
Injured leg	92.6 ±15.5	2.14 ±0.32	1.69 ±0.26	178 ±8	121.5+++ ±13.2	2.73+++ ±0.18	2.30+++ ±0.16	177 ±7	Left leg
2 legs[a]	113.4 ±20.3	2.87 ±0.45	2.36 ±0.36	188 ±6	145.7+++ ±10.6	3.41+++ ±0.23	2.87+++ ±0.21	189 ±8	2 legs

Significance: Patients uninjured cf. injured leg: *** $P<0.001$. Patients with cf. normals (1-leg and 2-leg), +++$P<0.001$
[a] $n=10$.

published (5) for young healthy men, and the mean difference in LV between the injured and uninjured legs of patients was closely similar to that found in a larger cross sectional anthropometric survey of patients entering the Joint Services Medical Rehabilitation Unit at Chessington (6). Theefore in terms of LV the data presented may be considered representative of patients recovering from leg fractures and healthy men so far measured in this laboratory.

In stationary bicycle ergometer tests care was taken to minimise the effects of learning and habituation to cycling (8) by allowing the subjects to practise and perform the complete series of exercise tests before definitive measurements were taken. Complete submaximal data were obtained on all subjects, but maximal experiments, as expected, were more difficult to conduct successfully. During heavy exercise patients often complained of muscle soreness, weakness, joint pain and expressed fears of re-injuring the affected limb. We felt it unwise to try and motivate patients beyond levels of exercise that they were prepared to go to voluntarily. However, of the 25 measurements of maximal performance attempted we did obtain successful observations on 15 patients but the final work load was only maintained for 1 min.

To achieve this a great deal of patience, and encouragement were required to gain the confidence of the patient and an understanding of what we were trying to achieve. To obtain a reliable duplicate measurement of maximal performance the test procedure was often repeated on 4 different occasions in each patient and thus our observations are open to the criticism that we may have been changing (through training) the parameter we were trying to measure. This we accept, though it should be noted that during daily repeated exercise studies on 2-leg work (8) no measurable change in $\dot{V}O_{2\,max}$ occurred during the first 4 days of observation. It is within the context of these facts that our results must be evaluated and interpreted.

During submaximal work in the patient, the most striking features are the decreased mechanical efficiency, the increased $f_{H\,1.5}$ and lower predicted $\dot{V}O_{2\,175}$ of the injured compared with uninjured limb. A lower mechanical efficiency and raised f_H at given $\dot{V}O_2$ for 1-leg work compared with 2-leg work are in accord with our previous findings on young adults (4, 5) but in all our previous studies of normal cycling we have been unable to demonstrate differences in pedalling efficiency even in most disparate groups of individuals irrespective of whether they have worked on (or seen) a bicycle before (see, e.g. 2). Even in the present study it should be noted that alhtough $f_{H\,1.5}$ is lower and $\dot{V}O_{2\,195}$ higher in the normal subjects compared with the patients in 2-leg work, the $\dot{V}O_{2\,900}$ is identical in the two groups (Table II). We have found (Davies and Sargeant, unpublished observations) that the difference in mechanical efficiency between injured and uninjured legs of the patients remains after an extended period of habituation to 1-leg bicycle ergometer exercise, but we now have some evidence that the effect may be due to the state of training of the injured limb (7). The raised $\dot{V}O_2$ for given $\dot{W}$ of the injured limb of patients is not reflected either by a similar change in $\dot{V}_{E\,1.5}$ or $V_{T\,30}$. Indeed

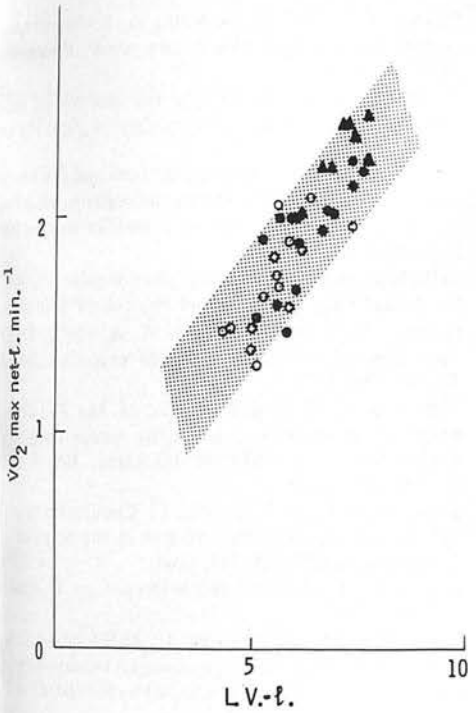

Fig. 1. Relationship of net maximal aerobic power output ($\dot{V}O_{2\ max\ net}$) of 1-leg to leg (muscle plus bone) volume. Patients: ●, uninjured limb; ○, injured limb; ▲, normal subjects. The shadowed area represents the limits (mean ±2 S.D.) previously found (4, 5) for healthy young subjects, age 18–27 years.

the $\dot{V}_{E\ 1.5}$ remains constant in patients and normal subjects independently of the type of exercise performed (Table II) though $V_{T\ 30}$ is higher in the normal subjects.

During maximal 1-leg and 2-leg exercise the patients reached similar $\dot{f}_{H\ max}$ values to the normal subjects (Table III) but $\dot{V}_{E\ max}$ values in both forms of work were significantly ($P<0.001$) lower. The diminished ventilatory response of the patients is probably a function of the time they were able to endure maximal effort. We have found previously (5) that the "$\dot{V}_{E\ max}$" can be increased by 30% in healthy well motivated subjects, who are prepared to sustain 1-leg exercise for 2–3 min at or above a level of work at which $\dot{V}O_{2\ max}$ occurs. The most surprising (and interesting) feature of the maximal performance study is that the $\dot{V}O_{2\ max\ net}$ of the patients injured leg is only 200 ml min^{-1} (10.6%) below that of their good leg. However, it should be noted that in turn the patients uninjured and injured 1-leg $\dot{V}O_{2\ max}$ and LV are 430 ml min^{-1} (18.8%) and 1.11 litres (15%); and 610 ml min^{-1} (25.6%) and 1.53 litres (21%) respectively lower than the right and left legs of the normal subjects (Table III). This suggests that as a result of leg fracture and subsequent hospitalization and immobilization, both limbs undergo a deterioration of physiological structure and function. It will be remembered that our patients were measured 173 days after sustaining their fractured limb and 70% of this time was spent either immobilized completely in bed or physically inactive because the patient was unable to support his own body weight. Clearly this enforced period of inactivity not only effects a deterioration in limb structure but is also likely to produce a degree of cardiovascular deconditioning (13). This would lend support to current practice in rehabilitation work that there is need for general conditioning exercises as well as those designed specifically to increase muscle strength and joint mobility.

The loss in function of uninjured and injured patients' legs is reflected in a reduced 2-leg $\dot{V}O_{2\ max\ net}$ of 510 ml min^{-1} (17.6%) when compared to their control subjects. The decline in 2-leg $\dot{V}O_{2\ max}$ is associated with the decrease in the uninjured and injured 1-leg $\dot{V}O_{2\ max}$ in patients, and the relationship of the 2-leg/1-leg $\dot{V}O_{2\ max}$ of the uninjured and injured leg lies within the limits expected for normal subjects (Fig. 2). However the regression lines for the injured and uninjured leg are significantly different, the former being displaced to the left (Fig. 2).

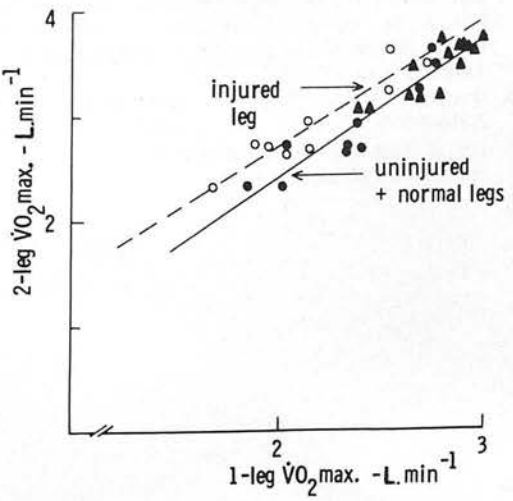

Fig. 2. The relationship of 1-leg to 2-leg aerobic power output ($\dot{V}O_{2\ max}$) in patients: ●, uninjured leg; O, injured leg. ▲ normal subjects.

These changes in 1- and 2-leg performance are undoubtedly, in part, a reflection of the different limitations to maximal effort in the two forms of work. In previous papers (4, 5) we have shown that maximal single limb exercise is mainly limited by the effective muscle mass which can be brought into play whereas when both legs are combined the ultimate limit is more likely $\dot{Q}$ and the ability of the circulation to transport O_2 and perfuse a given muscle mass with blood.

The present data support this concept: in 1-leg work the $\dot{V}O_{2\,max}$ of the patients is associated with a loss of limb muscle (plus bone) volume and in 2-leg work despite the reduced muscle mass of the injured leg the expected relationship (4, 5) of 2- to 1-leg $\dot{V}O_{2\,max}$ is maintained within normal limits (Fig. 2).

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Cardiopulmonary responses to exercise in obese girls and young women

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DAVIES, C. T. M., S. GODFREY, M. LIGHT, A. J. SARGEANT, AND E. ZEIDIFARD. *Cardiopulmonary responses to exercise in obese girls and young women.* J. Appl. Physiol. 38(3): 373–376. 1975.—A study of exercise performance was carried out in 17 obese girls and young adults. During submaximal steady-state bicycle exercise oxygen intake ($\dot{V}O_2$) for a given work output ($\dot{W}$) was raised in obese subjects but minute ventilation at a fixed carbon dioxide output, gas exchange, blood gases, and cardiac output at a given $\dot{V}O_2$ were similar to the values previously found for normals. In obese subjects high levels of $\dot{V}O_2$ for fixed $\dot{W}$ were also obtained on the treadmill but when these were standardized for body weight (unlike the bicycle test) it was shown that the obese girls and women exercised within the normal (expected) range of aerobic energy expenditure. During maximal performance the absolute $\dot{V}O_{2\,max}$ was the same in obese and nonobese subjects but for a given body weight, lean body mass, and leg muscle (plus) bone volume, $\dot{V}O_{2\,max}$ was reduced by 23.8, 16.3, and 24.5% respectively, in the former group. It was concluded that obesity though having minimal affect on responses to submaximal exercise is nevertheless associated with a marked reduction in physiological performance at or near maximal effort.

$\dot{V}O_{2\,max}$; females; obesity; body composition; cardiac output; performance

SEVERAL AUTHORS (see (1) for general review) have attempted to study the relationship between physiological performance and obesity, but their results are conflicting. Dempsey et al. (9) using male subjects during exercise on a stationary bicycle ergometer have shown that obesity is associated with a decrement in exercise performance particularly at maximal work levels, whereas Buskirk and Taylor (3) and Hansen (17) have found that they function normally. Indeed, both these latter studies suggest that fat per se does not impair physiological performance and must be viewed as a noncontributory load without effect on the capacity of either the cardiovascular system to transport or the working muscles to utilize oxygen. None of these studies has included children and female subjects.

We have studied the submaximal and maximal responses of young obese girls and women to exercise on a stationary bicycle ergometer and a motor-driven treadmill and related our finding to various indices of body composition.

MATERIAL AND METHODS

Through the good offices of the Weight Watchers Association, a group of volunteers was obtained of whom 17 were eventually studied in detail. The girls ranged from 6 to 25 yr, with 6 of them being under 13 yr old. They were of average height for their age, but 44% above their ideal weight (see Tanner et al. (21)). Their physical characteristics are given in Table 1 together with data for a match "control" group of nonobese females previously studied in our laboratories. All the girls were clinically examined before the studies and no abnormalities were found. They all understood the purpose of the study and agreed to participate as volunteers.

A simple progressive exercise test was carried out seated on an ergometer as described by Godfrey et al. (13) with the work load being increased every minute by 15-W increments for girls under 150 cm tall and by 20-W increments for taller girls. During this test, cardiac frequency (f_H) was measured from the electrocardiograph and ventilation ($\dot{V}E$) and O_2 consumption ($\dot{V}O_2$) were measured with a gasmeter and mixing chamber system using infrared CO_2 and paramagnetic O_2 analyzers, calibrated with several gas mixtures.

After a rest, the girls carried out a steady-state exercise test at approximately one-third and two-thirds of the maximal work output ($\dot{W}_{max}$) achieved in the simple progressive test. When a steady state of f_H, $\dot{V}E$, and $\dot{V}O_2$ had been reached (after 3–4 min) arterialized ear lobe blood was collected by the method of Godfrey et al. (15) and mixed venous Pco_2 was then measured by the plateau rebreathing technique (14). Cardiac output was calculated by the Indirect (CO_2) Fick method using the computer program of Godfrey (12).

After a further rest, the girls then carried out a simple progressive exercise test on a motor-driven treadmill analogous to that on the ergometer. In this test the girls walked at speeds varying from 1.9 to 7.1 $km \cdot hr^{-1}$ and the treadmill was elevated in increments of 2° every minute until the subject was unable to continue. During the test, f_H, $\dot{V}E$, and $\dot{V}O_2$ were measured as for the ergometer test.

Weight and height were measured and lean body mass was estimated after the method of Durnin and Rahaman (11), from the sum of four skinfold thicknesses (triceps, biceps, subscapular, and suprailiac). In addition, leg volume (muscle plus bone) was estimated using the physical anthropometric technique of Jones and Pearson (18). However, in eight cases we found it extremely difficult to obtain a reliable measure of skinfold thicknesses on the four leg sites which are needed to correct the total leg volume

for fat content, and so in these cases we have predicted the latter from the percent fat of the whole body using the following regression relationship obtained from the remaining nine obese females

$$\text{leg fat (\%)} = 3.36 + 1.25 \text{ body fat (\%)}$$
$$r = 0.84 \quad P < 0.001 \quad \text{SD} = 3.19\%$$

Maximal oxygen intake ($\dot{V}O_{2\,max}$). Previous experience had shown that subjects often reached their $\dot{V}O_{2\,max}$ on a standard progressive exercise test (7) and in the present study we tried to ensure that the elevation of the treadmill on the last two work loads was sufficient to obtain maximal performance. Subsequent analysis of our data revealed that 10 of the 17 subjects showed a "$\dot{V}O_2$ plateau"; final values of $\dot{V}O_2$ agreed within ±5% of each other. In the remaining seven subjects $\dot{V}O_{2\,max}$ was predicted from the $\dot{V}O_2/f_H$ relationship after the method described by Davies et al. (7) for children and Davies (5) for adults.

RESULTS

The physiological responses to steady-state exercise at approximately one-third and two-thirds of each subject's $\dot{W}_{max}$ are shown in Table 2.

For the obese children and young women, minute ventilation, tidal volume, blood gases, carbon dioxide, and cardiac outputs lie within the expected range for normal females and do not differ significantly from nonobese "control" values. However, for given work output (W) the $\dot{V}O_2$ is significantly higher ($P > 0.01$) for the obese compared with their nonobese counterparts (Fig. 1). For steady-state exercise the regression relationship for the obese is given by

$$\text{obese:} \quad \dot{V}O_2\ (l \cdot min^{-1}) = 0.459 + 0.0022\ (\dot{W}, \text{kpm} \cdot min^{-1})$$

The slope of the line is significantly different ($P < 0.001$) from that found previously for normal nonobese subjects.

TABLE 1. *Physical characteristics*

	Age yr	Wt, kg	Ht, cm	Σ4sf, mm	LBM kg	LV, l
Obese ♀	17.1	74.8	159.4	68.8	50.2	11.3†
(n = 17)	±5.5	±16.6	±11.2	±16.7	±10.0	±3.4
Nonobese ♀	17.7	51.0*	157.3	41.2*	38.2*	8.0*†
(n = 17)	±6.3	±11.1	±12.4	±12.1	±7.2	±2.3

Values are means ± SD. Σ4sf = sum of four skinfold thicknesses; LBM = lean body mass; LV = leg muscle (plus bone) volume. * Significance: $P < 0.001$. † n = 14.

Progressive exercise. For a given submaximal $\dot{V}CO_2$, $\dot{V}_E$ was the same in the progressive bicycle and treadmill exercise tests in the obese subjects. This was also true for V_T at a $\dot{V}_E$ of 30 $l \cdot min^{-1}$ and f_H at a $\dot{V}O_2$ 1.5 $l \cdot min^{-1}$. For the bicycle test the relationship of $\dot{V}O_2$ to $\dot{W}$ was similar to that given for the steady-state test (Fig. 1). During treadmill walking, the $\dot{V}O_2$ values of the obese subjects for a given speed and gradient was higher, as to be expected, than those for normal females. However, if the net O_2 cost (exercise $\dot{V}O_2$ − resting $\dot{V}O_2$) was expressed in terms of ml·kg (body weight)$^{-1} \cdot min^{-1}$ the difference between the two groups was removed.

At maximal levels of exercise the obese subjects reached higher absolute levels of $\dot{V}O_2$, $\dot{V}_E$, and f_H on the treadmill than the bicycle (all differencies were significant at or below the 5% level) and the absolute work output equaled or exceeded the expected value based on height given by Godfrey et al. (13) in 14 of the 17 females studied if allowance was made for the age of some of the girls. During treadmill exercise the mean $\dot{V}O_{2\,max}$ of the obese subjects was not significantly different from the expected "control" value (Table 3). However, in terms of body weight the $\dot{V}O_{2\,max}$ was significantly lower. The obese (30.7 ± 3.8 $ml \cdot kg^{-1} \cdot min^{-1}$) compared with 40.3 ± 5.8 $ml \cdot kg^{-1} \cdot min^{-1}$ in the nonobese ($P < 0.001$). This was also true (Table 3)

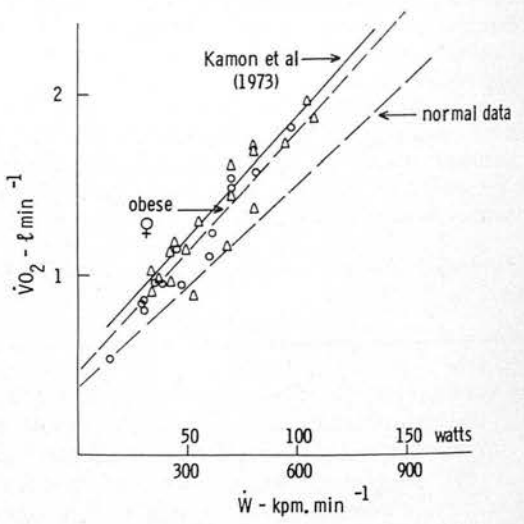

FIG. 1. Relationship between oxygen intake ($\dot{V}O_2$) and work output ($\dot{W}$) —— nonobese control data, —— obese females; —— line taken from Kamon et al. (19) using subjects with weighted legs (see text). ○, Young girls <18 yr; △, young women >18 years.

TABLE 2. *Steady state exercise*

		$\dot{W}$	$\dot{V}_E$, $l \cdot min^{-1}$	V_T, l	$\dot{V}O_2$, $l \cdot min^{-1}$	$\dot{V}CO_2$ $l \cdot min^{-1}$	P_{aCO_2}, mmHg	P_{aO_2}, mmHg	Q, $l \cdot min^{-1}$	f_H, beats·min^{-1}
Work I	Obese	35	24.5	0.71	0.95	0.76	35.9	88.8	10.2	139
		±10	±5.3	±0.20	±0.24	±0.25	±7.0	±3.3	±2.0	±14
	Control	40	23.4	0.68	0.89	0.77	35.5	93.0	8.8	146
		±11	±4.2	±0.19	±0.09	±0.14	±2.9	±5.9	±1.5	±16
Work II	Obese	70	40.0	1.08	1.40*	1.30	34.7	88.7	11.9	166
		±21	±7.5	±0.37	±0.34	±0.35	±5.8	±3.4	±1.4	±10
	Control	80	41.5	0.98	1.26	1.34	36.9	96.1†	10.7	179
		±21	±9.2	±0.29	±0.28	±0.33	±1.7	±2.6	±2.0	±11

Values are means ± SD. Significance: * $P < 0.05$. † $P < 0.001$.

when $\dot{V}O_{2\,max}$ was expressed in terms of LBM (44.4 ± 6.5 obese, 53.0 ± 6.9 ml·kg^{-1}·min^{-1} nonobese, $P < 0.001$) and estimates of limb muscle (plus bone) volume (188 ± 21 obese, 249 ± 25 ml·l(LV)$^{-1}$·min^{-1} nonobese $P < 0.001$). The latter relationship of $\dot{V}O_{2\,max}$ to LV is shown graphically in Fig. 2.

The regression relationship includes the predicted values of $\dot{V}O_{2\,max}$ (see Fig. 2 legend). All the points for the obese lie below the expected mean line for normal women, which gives rise to a parallel displacement of the calculated regression equation in the obese group.

DISCUSSION

The study was conducted with the aim of comparing the physiological responses to submaximal and maximal exercise of obese girls and young women, particularly with reference to certain indices of body composition. The results suggest that during maximal exercise there is a marked decrement in exercise performance in obese females compared with their matched "controls" but during light and moderate exercise their metabolic (with the exception of $\dot{V}O_2$ at given $\dot{W}$) and cardiovascular responses are entirely normal. We have found no evidence to suggest that ventilation and cardiac output are affected by obesity during submaximal exercise either on a stationary bicycle or motor driven treadmill. Minute ventilation rises to meet the requirements of CO_2 elimination, blood gas tensions are maintained and, for a given $\dot{V}O_2$, Q lies within the normal range expected for normal nonobese females (Table 2). Thus our data are in close agreement with those of Dempsey and his colleagues (9).

TABLE 3. *Maximal oxygen intake* ($\dot{V}O_{2\,max}$)

	$\dot{V}O_{2\,max}$			
	l·min^{-1} Abs	ml·kg^{-1}min^{-1} (Wt)	ml·kg^{-1}·min^{-1} (LBM)	ml·l^{-1}·min^{-1} (LV)
Obese (n = 17)	2.27 ±0.52	30.7 ±3.8	44.4 ±6.5	188† ±21
Nonobese (n = 17)	2.03 ±0.45	40.3* ±5.8	53.0* ±6.9	249*† ±25

Values are means ± SE. * Significance: $P < 0.001$. † n = 14.

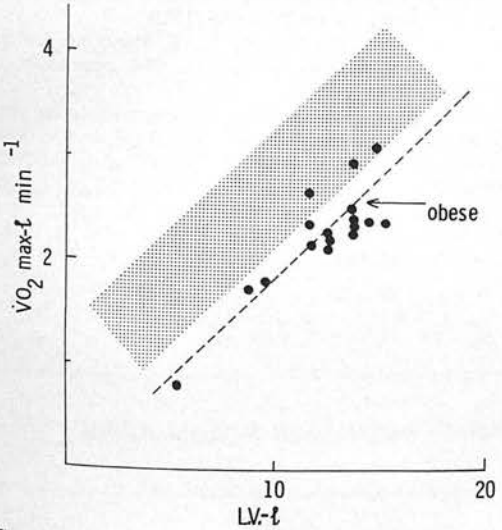

FIG. 2. Relationship between maximal aerobic power output ($\dot{V}O_{2\,max}$) and estimates of limb muscle (plus bone) volume (LV) in obese females. Shaded area represents limits previously found for normal nonobese girls (7).

Our observation of an increased $\dot{V}O_2$ for given work output in cycling compared with walking agrees with the data presented by Parizkova (20) and Dempsey et al. (9) but not apparently with Hansen (17). He found normal metabolic costs on both the treadmill and ergometer in volunteers who became obese experimentally, but they were only 15–19% overweight which is considerably less than our subjects (Table 1). It would seem that whether weight is carried either as excess fat (as in the present investigation) or as an external load (16) the aerobic energy expenditure is proportional to the total weight carried. During walking there is no evidence to suggest that the working muscles of these subjects are any less (or more) efficient at consuming energy than their normal (thinner) counterparts. The increased aerobic cost of cycling must be judged in this light.

Dempsey et al. (10) have suggested that the increased oxygen cost of breathing reported by some authors (e.g., 2) is unlikely to be a factor in the excessive rise of $\dot{V}O_2$ with $\dot{W}$ during weight supported work on a bicycle ergometer in the obese. In this we agree since as we have seen pulmonary function and blood gas tensions are normal in our investigation. Dempsey et al. (10) state that the increase in $\dot{V}O_2$ was more likely to be related to the increased postural component of work and point out that fat subjects often find it necessary to rotate the trunk and buttocks in order to facilitate the movement of the thighs about the saddle during cycling. We observed this phenomenon in a few of our young women, but if increased postural effort was the cause of the observed rise in $\dot{V}O_2$ we would have expected from previous experience (8) the relationship between $\dot{V}O_2$ and $\dot{W}$ to become curvilinear at or near maximal work levels and this is clearly not the case (Fig. 1). Nevertheless it would be dangerous for us to extrapolate from one type of work to another, and Dempsey's theory cannot be discounted. However, there is one other possible explanation which must be briefly considered. Cotes (4) has suggested that the additional cost of pedaling the bicycle in overweight subjects might be due to the extra work required to move their larger leg mass. This is an interesting theory, but if it were so one would expect to be directly related to leg volume. In the present study the association between $\dot{V}O_2$ and LV is significant ($P < 0.001$, $r = +0.67$), but if weight is held constant the relationship between the two variables disappears. Further one would expect as found by Kamon et al. (19), who attached weights to the legs of normal subjects to increase their effective leg mass during cycling, that the $\dot{V}O_2/\dot{W}$ line would show a parallel displacement to the left of the normal regression relationships. In other words the work of overcoming inertia and providing momentum to overweight limbs would be accounted for in the energy necessary to pedal the bicycle at constant frequency against zero load (the intercept of the $\dot{V}O_2/\dot{W}$ line) and clearly this is not so in

our data. The slope of the $\dot{V}O_2/\dot{W}$ line is significantly ($P < 0.001$) different from that found for normal subjects.

Maximal exercise. Buskirk and Taylor (3) have described fat as an inert noncontributory load which does not impair maximal performance. They base their suggestion on the observation that if $\dot{V}O_{2\,max}$ is standardized for fat-free body weight in obese subjects it lies within the normal range. Certainly we have found this to hold for normal men, some of whom were overweight (6), but clearly in light of the present results such views are no longer tenable for the grossly obese. In absolute terms the $\dot{V}O_{2\,max}$ of our subjects were compatible with nonobese controls, but for given LBM and LV, $\dot{V}O_{2\,max}$ was reduced by 16 and 24%, respectively. Indeed the line relating $\dot{V}O_{2\,max}$ to LV (Fig. 2) is very similar to that found prior to the commencement of a rehabilitation programme in patients who have been grossly inactive due to immobilization (Davies and Sargeant, unpublished observations). Certainly fat may act (as in treadmill walking) as an inert mass but during maximal exercise it is tempting to suggest that carriage of fat for prolonged periods has played some indirect role in the etiology of the relative effort intolerance of the obese. The data from the present study provide no direct evidence on this point; we can only conclude that maximal exercise performance related to fat-free indices of body size is impaired in obese young women while their cardiopulmonary responses to a given submaximal metabolic stress are normal. Taken together these results might indicate that weight reduction and an increased physical activity are a prerequisite for a return to normal maximal aerobic function, but further research is required before the precise contribution of these two important factors can be elucidated.

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Circadian variation in physiological responses to exercise on a stationary bicycle ergometer

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Circadian variation in physiological responses to exercise on a stationary bicycle ergometer

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Davies, C. T. M. and Sargeant, A. J. (1975). *British Journal of Industrial Medicine*, **32**, 110-114.
Circadian variation in physiological responses to exercise on a stationary bicycle ergometer. The responses of six healthy male subjects to submaximal and maximal exercise on a stationary bicycle ergometer have been investigated over a 24-hour period. Measurements were made on each subject at approximately three-hourly intervals and they included minute ventilation at a carbon dioxide output of 1·5 l min^{-1} ($\dot{V}_{E\,1\cdot5}$), tidal volume at a fixed $\dot{V}_E$ of 30 l min^{-1} ($V_{T\,30}$), oxygen intake ($\dot{V}O_2$) at a work load ($\dot{W}$) of 150 W ($\dot{V}O_{2\,150}$), tympanic temperature (T_{ty}) and cardiac frequency at a $\dot{V}O_2$ of 1·5 l min^{-1} ($f_{H\,1\cdot5}$). The experiments were conducted in three parts: on the first occasion two subjects were measured during exercise; on the second occasion a further four subjects were observed in a similar way but starting from a baseline of zero load, and the measurements also included an estimate of cardiac output ($\dot{Q}$) using a rebreathing technique. Finally the maximum aerobic power output ($\dot{V}O_{2max}$) was measured in three of the subjects in early morning and late evening. Diet and habitual physical activity were held constant between the exercise test on all three occasions.

The results show that in the first two subjects $f_{H\,1\cdot5}$ and T_{ty} had a rhythmic pattern of variation with time of day whereas $\dot{V}_{E\,1\cdot5}$, V_{T30}, and $\dot{V}O_{2\,150}$ remained fairly constant. The variation in $f_{H\,1\cdot5}$ was associated with T_{ty}; the two variables reached a minimum at ~0500 hr and a maximum at ~1200 hr. These results were confirmed on the remaining subjects but the changes in $f_{H\,1\cdot5}$ and T_{ty} were shown to be more variable and reduced in magnitude. Further, if the changes were calculated from a baseline of zero load, it was shown that the absolute changes observed in $f_{H\,1\cdot5}$ and T_{ty} were not due to the exercise per se but to changes in the basal level from which each subject operated. In addition it was shown that $\dot{V}O_{2max}$ and $\dot{Q}$ remained constant and were independent of the time of day.

It is concluded that provided the exercise test conditions are rigidly standardized and subjects exercise from a controlled baseline there is no evidence for circadian variation in the change of responses to work at submaximal or maximal effort.

There is a growing volume of literature on the effects of circadian rhythms on the physiological performance of man at rest, during sleep, and while engaged in various physical activities (see Kleitman (1949) and Mills (1966) for general review) but to our knowledge no comprehensive study has been undertaken of the effects of time of day on physical work output and exercise tolerance. Many authors have noted changes of body temperature and cardiac frequency at rest (Conroy and Mills, 1970) and have presented data on athletic performance at two or more different times during the day (Conroy and O'Brien, 1973; Wahlberg and Åstrand, 1973) but no detailed study of the responses to submaximal and maximal exercise throughout a 24-hour period has been attempted.

In the present investigation six healthy male subjects have been studied during work on a stationary bicycle ergometer at three-hourly intervals throughout a day and the following night. A preliminary report on the first two subjects measured has already been given (Crockford and Davies, 1969).

Material and methods

The six subjects were required to attend the laboratory at 0800 am in the postabsorptive state. Their physical characteristics are given in Table 1. The experiments were

TABLE 1

PHYSICAL CHARACTERISTICS OF THE SIX SUBJECTS

Subject	*Age (yr)*	*Height (m)*	*Weight (kg)*
GWC	35	1·74	74·5
CTMD ..	35	1·78	73·9
SR	21	1·64	62·0
AL	32	1·73	75·4
MP	30	1·80	81·0
MB	32	1·77	69·0
Mean	30·8	1·74	72·6
±SD	±5·2	±5·4	±6·5

conducted in three parts. On the first occasion two subjects (GWC and CTMD) were studied during submaximal exercise at regular intervals over a 27-hour period. On the second occasion the remaining four subjects were similarly studied, and on the final occasion the maximal aerobic power of three of the subjects were measured at 0800 hr in the morning and 1800 hr in the evening of the same day. Throughout each experimental period attempts were made rigidly to standardize the diet and physical activity of each subject. The following routine was carried out: the subjects were measured sequentially on the bicycle ergometer. A standard progressive submaximal test was given to each subject at three-hourly intervals in which the work load was raised every 5 minutes. At least five work loads were performed, the aim being to span the whole range of the subjects' submaximal work capacity. In the first pair of subjects exercise was started at 50 W but in the last four subjects, who were studied on a separate occasion, a baseline of zero load was used. This enabled changes in exercise performance to be calculated (Davies and Crockford, 1971; Whipp and Wasserman, 1972). On finishing the bicycle test, the subject was allowed to eat a measured portion from a standard diet which was prepared in advance. The diet was so arranged that the food was equivalent to a 24-hour energy intake of 11 220 kJ, and each intake after exercise was equivalent to~1420 kJ. The food contained 65% carbohydrate, 20% fat, and 15% protein, and care was taken to offer the subject as much variety as possible in small portions. A fluid intake of 25 ml, which if not water was accounted for in the calorie balance, was allowed. Following the exercise the subjects were allowed to rest or read or listen to the radio and to take light exercise (walk around, play table tennis, etc). The light exercise periods were not allowed to exceed 30 minutes of the total time between each definite bicycle ergometer test. Immediately before the beginning of the exercise test the subject voided his bladder.

During exercise oxygen intake ($\dot{V}O_2$) and pulmonary ventilation ($\dot{V}_E$) were measured using a standard open circuit technique (Davies and Shirling, 1967), and body core temperature (T_{ty}) was monitored from a thermistor placed close to the tympanic membrane. The thermistor was heavily insulated from the outside environment and was kept in situ throughout the 24 hours of the experiment. Cardiac frequency (f_H) was measured using a differential electrocardiograph amplifier and instantaneous ratemeter, and cardiac output was estimated in four of the six subjects using a CO_2 rebreathing technique (Jones *et al.*, 1967). In the event of an unsuccessful plateau $P\dot{V}CO_2$ was estimated by extrapolation. No correction was made for the downstream phenomenon, arterialized blood P_{CO_2} was not taken, and cardiac output ($\dot{Q}$) was computed using a program devised by Godfrey (1970) assuming a normal dead space for each subject. Further details and discussion of the limitations of the method for measuring $\dot{Q}$ are given by Godfrey and Davies (1970) and Godfrey *et al.* (1971).

Maximal aerobic power ($\dot{V}O_{2\,MAX}$) was measured using a continuous exercise test in which the subject started at approximately 90% of his maximal work level predicted from his previous submaximal responses. The criteria for maximal performance were that $\dot{V}O_2$ showed no further increase with increasing work load (W) and plateau values of $\dot{V}O_2$ agreed within ±5% (Davies, 1968).

Results

The results collected on the first pair of subjects (GWC and CTMD) showed that $f_{H1\cdot5}$ and T_{ty} varied with the time of day whereas $\dot{V}O_2$ at a fixed rate of work, $\dot{V}_{E\,1\cdot5}$, and $V_{T\,30}$ remained fairly constant. The variations in $f_{H\,1\cdot5}$ and T_{ty} were associated ($r = +0{\cdot}47$, $P < 0{\cdot}001$); both variables reached a minimum at 05.00 hr followed by a gradual rise throughout the morning to reach a maximum between 14.00 and 18.00 hr. The rise in T_{ty} and in $f_{H\,1\cdot5}$ was of the order 0·8° C and 14 beats min^{-1} respectively. These results were confirmed by observations on the remaining subjects during phase 2 of the study but the change in T_{ty} and $f_{H\,1\cdot5}$ was shown to be more variable and reduced in magnitude (Fig. 1). In subject AL, T_{ty} rose sharply in the morning, reaching a maximum at 12.00 hr, remained fairly constant during the day, and then fell sharply in the night, whereas the T_{ty} in SR, MB and MP varied more rhythmically. In these three subjects, a T_{ty} minimum was reached at 05.00 hr and a maximum between 15.00 hr and 17.00 hr. The inter-subject variability of $f_{H\,1\cdot5}$ was less than for T_{ty}; $f_{H\,1\cdot5}$ rose in the morning and fell during the night, but in contrast to the results on CTMD and GWC no well-defined maximum was observed (Fig. 1). The mean differences between day and night time $f_{H\,1\cdot5}$ and T_{ty} values for the six subjects were 0·76° C and 7 beats min^{-1} respectively.

The results shown in Fig. 1 indicate the absolute changes of $f_{H\,1\cdot5}$ and T_{ty} during exercise with time of day but from these data it is not possible to ascer-

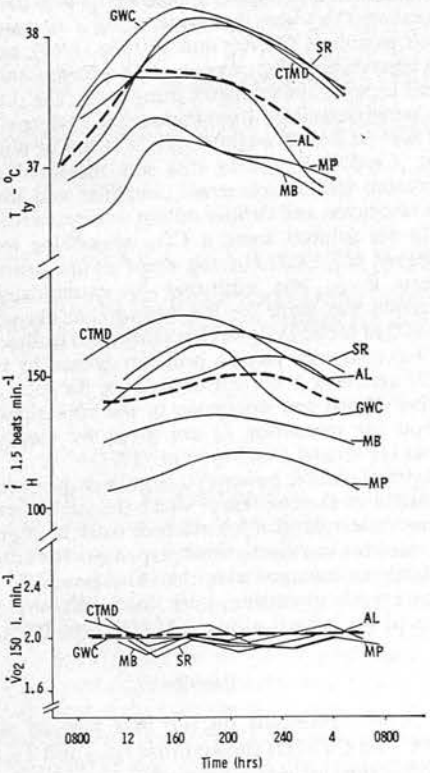

FIG. 1. Absolute changes in $f_{H\ 1\cdot5}$, T_{ty}, and $V_{O_2\ 150}$ with time of day. Individual and mean (– – –) values for six subjects. T_{ty} represents the aural temperature recorded at the end of 20 minutes' continuous submaximal test. They may not be taken as representative steady state values of T_{ty} which usually require 45 minutes of exercise to establish due to the high thermal capacity of the body at the onset of work.

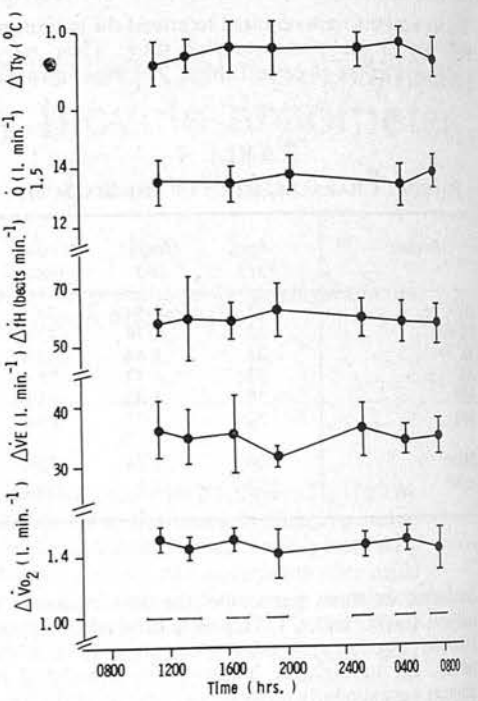

FIG. 2. Changes in exercise tympanic temperature (ΔT_{ty}), cardiac frequency at given $\dot{V}o_2$ of 1·5 l min^{-1} (Δf_H), minute ventilation at given $\dot{V}co_2$ of 1·5 l min^{-1} ($\Delta\dot{V}_E$), and oxygen intake at given workload of 150 W ($\Delta\dot{V}O_2$) measured from a baseline of zero load and cardiac output at given $\dot{V}o_2$ of 1·5 l min^{-1}. Mean±SD data for four subjects. The bars at the bottom of the figure indicate the mean ±SD of the times during which the measurements were taken.

tain whether they reflect a genuine circadian variation in submaximal exercise performance or whether they are merely a consequence of a change in the physiological baseline from which each subject was operating. In subjects CTMD and GWC it was no possible to answer this question since the first work load performed by each subject was 50 W but in the second series of experiments on four subjects the measurements were repeated from a baseline of

TABLE 2

RESPONSES TO MAXIMAL EXERCISE MEASURED IN THREE SUBJECTS AT 0800 HR AND 1800 HR ON THE SAME DAY

Subject	0800 hours			1800 hours		
	$\dot{V}_{E\,max}$ (*l min*$^{-1}$)	$\dot{V}_{O_2\,max}$ (*l min*$^{-1}$)	$f_{H\,max}$ (*beats min*$^{-1}$)	$\dot{V}_{E\,max}$ (*l min*$^{-1}$)	$\dot{V}_{O_2\,max}$ (*l min*$^{-1}$)	$f_{H\,max}$ (*beats min*$^{-1}$)
MB	166·48	4·58	174	155·39	4·36	179
AL	145·01	3·71	184	150·62	3·75	180
SR	130·34	3·14	188	106·78	2·90	188
Mean	144·27	3·81	182	137·60	3·67	182
SD	±18·18	±0·73	±7	±26·79	±0·73	±5

pedalling against zero load. In addition, cardiac output, and at a later stage in three of the subjects, maximal aerobic power were measured. The data for submaximal exercise are shown in Figure 2. It will be seen that $\Delta\dot{V}O_2$ for given $\dot{W}$ and $\dot{V}_E$ remain unchanged throughout the period of the study. Calculating the results from a baseline of zero load also obliterates the variation in T_{ty} previously observed and markedly reduces the rise in f_H. Cardiac output for a given $\dot{V}O_2$ remains constant during the 24 hours of the experiment.

Maximal performance

The responses to maximal exercise measured in the early morning and late evening are shown in Table 2.

The mean maximal cardiac frequencies were identical during the two periods of the day; $\dot{V}O_{2max}$ and $\dot{V}_{Emax}$ were 0·14 l min^{-1} and 6·7 l min^{-1} lower during the evening but none of these differences was statistically significant.

Discussion

The initial experiments on two healthy male subjects (CTMD and GWC) were designed to evaluate the responses to submaximal exercise in relation to time of day, with diet and habitual activity held constant. They were repeated on a further four subjects during phase 2 of the investigation. The results show that both the circulatory and thermal responses to work vary with time of day during a 27-hour period but the periodicity and magnitude of the changes vary for each subject (Fig. 1). This gave rise to a large intersubject variability for both $f_{H1·5}$ and T_{ty}. The average cardiac frequency and tympanic temperature were 7 beats min^{-1} and 0·76° C higher in the day than the night. These changes were observed against a background of metabolic constancy (Fig. 1): $\dot{V}O_{2\,150}$, $\dot{V}_{E\,1.5}$, and $\dot{V}_{T30}$ remained constant throughout the investigations and were independent of the time of day. These results agree closely with the observations of Klein, Wegman, and Brüner (1968) and the recent report of Wahlberg and Åstrand (1973). The latter authors found an unchanging aerobic cost of work but a significant ($P < 0·05$) increase in f_H of 4 beats min^{-1} at 100 W (approximately equivalent to a $\dot{V}O_2$ of 1·5 l min^{-1}) during the day (15.00 hr) compared with the night (03.00 hr) time. However, these changes are small and in our experiments no account was taken of the possible confounding influence of change in basal level from which each subject was operating during exercise. In addition, though the changes in f_H at a given $\dot{V}O_2$ gave rise to a change in predicted maximal aerobic power ($\dot{V}O_{2max}$), no direct measurements of this latter parameter were made in our investigation, and it is unclear whether the changes in f_H would be reflected in comparable changes of $\dot{Q}$. Therefore in the second series of experiments, we repeated the exercise studies from a baseline of constant pedalling against zero load and included measurements of $\dot{Q}$ using a rebreathing technique. In the final experiments we made direct measurements of $\dot{V}O_{2max}$ in three of these four subjects in the early morning and late evening. The data from these experiments show that $\dot{Q}$ at a given $\dot{V}O_2$, and $\dot{V}O_{2max}$ remained constant (cf Wahlberg and Åstrand, 1973) and are independent of the time of day (Fig. 2; Table 2). Further, calculating the f_H and T_{ty} data from a baseline of zero load obliterates variations in circulatory and thermal responses previously observed (Fig. 1).

These results taken together are important and, contrary to our previous expectation (Crockford and Davies, 1969), suggest that provided conditions are standardized, measurements taken from fixed baseline exercise performance remain the same throughout a given 24-hour period. The observed changes in absolute f_H and T_{ty} must be due to some underlying basal circadian rhythm and not to the response to exercise *per se*. What therefore is the nature of this underlying rhythm which affected our earlier results? The answer may lie in our observations of $\dot{V}O_{2\,150}$, $\dot{Q}$, and $\dot{V}O_{2max}$ in the first two and third stages of our study respectively. It will be noted that these three variables remain constant irrespective of whether they are calculated in absolute or relative (Δ) terms. This is in contrast to what has been found for subjects who have been allowed to sleep during the night (Conroy and Mills, 1970). Under these conditions $\dot{V}O_2$ shows a marked fall during the somnambulant periods. The constancy of $\dot{Q}$ and the absolute changes in f_H imply mathematically (but not necessarily biologically) a small rhythmic change in basal cardiac stroke volume with time of day, which in turn (again by implication from our first series of experiments) must be associated with changes in T_{ty}. Provided posture and the effective muscle mass performing the work remain fairly constant, as in the present experiments which involved upright two-leg work on a bicycle ergometer, there is some common agreement (Bevegård and Shepherd, 1967) that a change in stroke volume usually implies a displacement of blood to the periphery, possibly to subserve the requirements of thermoregulation. Indeed, Kaneko, Zechman, and Smith (1968) have shown that rhythmic changes in peripheral blood flow do occur. They observed that arm blood flow measured by venous occlusion plethysmography reaches a maximum in the later afternoon or early evening which corresponds with our circulatory observations. Thus one is led to the conclusion that the changes in f_H we originally observed must be thermogenic and not metabolic in origin.

A change in T_{ty} can only result from an increase in the dissipation or conservation of body heat. Several

authors (Mills, 1966) have noted a rhythmic change in skin temperature (Ts), the curve relating to time of day being the mirror image of that found to T_{ty}. In a previous study (Crockford, Davies, and Weiner, 1970) we also noted a rise in Ts during the night but a maximum in the T_{ty} at sweat onset during the day. Taken together all these data and those of the present investigation suggest that variations in heat loss mechanisms, particularly the interaction between skin, blood flow, and sweat onset, may be more important than heat production in regulating the rhythmic basal changes in observed f_H and T_{ty}. In exercise these factors will be operative, but our data merely indicate that they will give rise to changes of absolute f_H and T_{ty} of the same order as those seen during baseline exercise during which no external work is performed. Thus in practical terms, particularly with reference to circadian variation of performance, they will have little bearing. Aerobic power output and physiological response to a given change in work output during exercise on a stationary bicycle ergometer remain constant and independent of the time of day.

We wish to thank Mr. G. W. Crockford for his active participation in the early experiments and Mr. Stephen Riggs for technical assistance. We are deeply indebted to the four subjects who took part in the second series of experiments for their wholehearted co-operation and cheerfulness in the face of stringent demands.

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PHYSIOLOGICAL PERFORMANCE AND WORK CAPACITY OF SUDANESE CANE CUTTERS WITH *SCHISTOSOMA MANSONI* INFECTION

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Abstract. Physiological tests of work performance and measurements of field productivity were made in 194 Sudanese cane cutters in order to study the effect of *Schistosoma mansoni* infection. The cane cutters were selected from two age ranges (16–24 and 25–45 years) and subdivided into three clinical groups: not infected, infected with, and infected without clinical signs of hepatosplenomegaly. Men infected with *Schistosoma haematobium*, malaria (blood film), or with hemoglobin levels less than 10 g/100 ml were excluded. There was a statistically significant ($P < 0.002$) higher mean hemoglobin concentration in those not infected, but the mean difference was less than 1 g/100 ml. Submaximal responses to exercise on a stationary bicycle ergometer, oxygen intake, ventilation, tidal volume, cardiac frequency and estimated maximal aerobic power output calculated both in absolute terms and relative to lean body mass and leg volume were similar in the six groups of cane cutters. No significant differences were found in physique, body composition or in thermoregulatory function tests. The cane cutters were found to have little natural acclimatization to heat in terms of sweating capacity when compared with a group of fully acclimatized Sudanese soldiers. The mean productivity (mean daily weight of cane cut per man) was significantly correlated with the individual's estimated maximum aerobic capacity determined in the laboratory, but not with the degree of *S. mansoni* infection. The noninfected group was less "efficient" (mean productivity:oxygen intake) during cutting than the infected groups but a larger proportion of the noninfected were in their first season of cutting. There was a positive correlation between the number of seasons' cutting experience and the individual's age, degree of infection and mean productivity. Cane cutters studied in this investigation were a relatively fit, active population from whom the more seriously ill were excluded. These results do not, therefore, necessarily reflect the effects of *S. mansoni* on physiological work capacity and productivity of more static populations in areas of high endemicity.

INTRODUCTION

In the early stages of schistosomal infection, fatigue and debility are symptoms commonly described in all age groups. It is also widely believed that the chronic ill-effects of the disease profoundly affect growth, physical working capacity, and productive output of infected populations.[1–4] There have, however, been few objective studies which have attempted to define the nature of the incapacity to work and the extent to which the disease influences physiological performance. Information available on these aspects is at present conflicting and controversial. Extreme fatigue and physical exhaustion after short bouts of physical exercise has been described in infected Nigerian children,[5] but in controlled studies the physiological response to exercise in East African[6] and Bantu[7] school-children with *Schistosoma mansoni* infection was found to be unimpaired. The results of a recent investigation on young adult males[8] suggest, however, that some physiological parameters of physical fitness are affected by *S. mansoni* and significantly improved after treatment.

In Egypt, where the prevalence of both *S. mansoni* and *S. haematobium* is high, the loss of labor output among 14 million infected people is estimated to be as much as 35%,[9] and in mainland China, where the more pathogenic *S. japonicum* is

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endemic, an average loss of 40% of an adult's capacity to work has been reported.[4] The difficulties involved in assessment of the socioeconomic impact of schistosomiasis through the (presumed) loss of working capacity have been well documented.[10,11] This applies particularly to primitive communities where continuous industrial employment is not the norm, the method of work generally is uncontrolled, and where intercurrent disease is usually a complicating factor. From the evidence available, a WHO Expert Committee was thus led to conclude that "it is still not possible to compute in economic terms the precise extent of the damage caused to communities by schistosomiasis and that therefore rigorous and economic analysis of the benefits of control was not possible."[12]

In the present investigation a more direct approach has been adopted in order to measure working capacity in a selected population with *S. mansoni* infection. An attempt has been made to assess the effect of *S. mansoni* on physiological tests of work performance and heat tolerance under laboratory conditions, and to relate the findings to productive output in the same group of men performing a self-paced task under natural working conditions. The group of men selected for these studies were Sudanese cane cutters living in two villages in the Guneid sugar plantation on the Blue Nile.

MATERIALS AND METHODS

Location

Guneid is a small township situated approximately 100 km southeast of Khartoum on the east bank of the Blue Nile and facing the Gezira irrigated area to the west. It is important as a center for the Sudanese sugar industry, with a large refinery normally in production from November to April. Over 20,000 acres are planted with cane in the immediate area of the refinery on land partly worked by tenant farmers and supplemented by a migrant labor force of about 1,000 cane cutters. During January and February, when the investigations were made, daily sunshine for the area averaged 10 to 11 hours per day, rainfall was zero, wind speed 1 to 5 meters/second, relative humidity 32 to 44%, and dry-bulb temperature in the shade was 34 to 36° C (maximum), 15 to 16° C (minimum).

Procedure

The investigations were divided into four stages: 1) a preliminary clinical and parasite screening of the cane cutters was undertaken in December 1973 and January 1974 in order to identify infected and noninfected groups; 2) laboratory tests of working capacity and heat tolerance as well as assessment of physique were made in January 1974 on 194 men who were divided into groups after rescreening for schistosomiasis, malaria and other parasitic infections; 3) the individual studies on productivity in the cane fields were made in February 1974 on the same men selected for laboratory tests. The men were given free opportunity to cut cane to the limit of their capacity, with and without motivation by a bonus system. Physiological observations of energy expenditure, heart rate levels and sweat loss were made during the field studies; 4) finally, an ancillary experiment was made in Khartoum and at Guneid in June 1974 involving the intensive heat acclimatization of a group of volunteers. This was designed to provide a scale against which the observed degree of heat tolerance in the cane cutters could be compared.

The procedures were demonstrated beforehand to all the cane cutters who took part, and it was explained that any man could withdraw from the experiment at any time if he wished. On completion of the field studies, arrangements were made for medical treatment to be available, if required, at the local hospital, and a significant proportion of the men were subsequently given treatment with niridazole for schistosomiasis.

Screening and Selection

In the preliminary screening, about 500 adult cane cutters between the ages of 16 and 60 years were clinically examined in their villages at Guneid by a team from the Faculty of Medicine, University of Khartoum. Blood films were examined for malarial parasites, urine for schistosome eggs, and egg counts (eggs/g) were made on at least two consecutive fecal specimens by the Kato thick smear method.[13] In recent studies,[14] morbidity has been assessed by quantitative egg excretion during a 4-year period but there is less evidence of a high degree of correlation between symptoms and fecal egg counts in short term studies.[15] It was therefore decided to group the cane cutters into noninfected (no egg counts) and

TABLE 1

Mean environmental temperatures (°C ± S.D.) inside and outside (shade) the laboratory at Guneid during January 1974

Time (hr)	Place	Dry bulb	Wet bulb	Globe temperature	WBGT index*
0700	Inside	23.5 ± 1.3	13.8 ± 1.5	29.4 ± 2.0	15.8 ± 1.5
	Outside	17.1 ± 2.4	10.9 ± 2.1	25.4 ± 2.1	12.5 ± 1.9
1500	Inside	28.7 ± 1.1	16.1 ± 0.9	33.3 ± 3.8	18.8 ± 1.0
	Outside	32.4 ± 2.6	17.3 ± 1.4	37.9 ± 6.3	20.8 ± 1.7

* See reference 16.

infected (egg counts in any number) with and without clinical signs of hepatosplenomegaly. The assumption was made that, in the absence of malaria and other parasitic diseases, cane cutters with hepatosplenomegaly were more seriously affected by *S. mansoni* infection. Some reservations must, however, be placed on this method of classification for, although the migrant cane cutters did not originate from highly endemic malarious areas, a proportion of them may have had malarial splenomegaly in the absence of parasites in blood films. Those shown to be infected with *S. haematobium* alone or intercurrently were excluded, as were cane cutters found by blood and stool examinations to have malaria or other parasitic infections or to have hemoglobin levels less than 10 g/100 ml. Finally, 194 men were selected and divided into six groups: (*A*) noninfected (16–24 years of age); (*B*) intestinal, with *S. mansoni* eggs in stools (16–24 years of age); (*C*) intestinal with eggs and clinical signs (16–24 years of age); (*D*) not infected (25–45 years of age); (*E*) intestinal (25–45 years) and (*F*) intestinal with signs (25–45 years). None of the men with clinical signs had gross hepatosplenomegaly or ascites.

Laboratory Measurements

Each man attended, for 1 day, a laboratory (partially air-conditioned) in Guneid and was paid an equivalent day's wage for taking part in the experiments. For the purpose of these tests, between 6 and 12 men chosen at random from the six groups were transported from the villages each day. Before the investigations the men were provided with a meal and a re-examination was made for malarial parasites and hemoglobin concentration (Sahli). Hospital "Identabands" were fastened to the wrist and the bands were retained in order to facilitate identification during the subsequent field studies. With the assistance of an interpreter, each man completed a sociological questionnaire which provided details of family and occupational history.

Climatic conditions recorded daily inside and outside the laboratory during the period of the tests are given in Table 1.

Anthropometry. Body weight and height were measured using a balance accurate to ±5 g and a stadiometer. Skinfold thickness was recorded at four sites—biceps, triceps, subscapular and suprailiac, and lean body mass was estimated from skinfold thickness and body weight by the method of Durnin and Rahaman.[17] Leg muscle (plus bone) volume was estimated from measurements of height and the circumferences of thigh and calf.[18]

Physiological response to exercise. Prior to the definitive test on a stationary bicycle ergometer, each subject was given a 15-minute practice period. The exercise was in the form of a progressive test, the work rate being raised every 3 minutes by approximately 300 kpm min^{-1} from zero load to the limit of the individual's work capacity.[19] Minute ventilation, oxygen intake and cardiac and respiratory frequencies were measured during the final minute of each work load. Samples of expired air were taken from the mixing chamber for analysis of CO_2 (Katherometer, Cambridge Instruments Ltd.) and O_2 (paramagnetic analyser, Servomex Ltd.). The data from the exercise test were expressed for each subject in terms of oxygen intake ($\dot{V}O_2$) at a work load of 900 kpm min^{-1} ($\dot{V}O_{2\,900}$); minute ventilation ($\dot{V}_E$) at a CO_2 output ($\dot{V}CO_2$) of 1.5/1 min^{-1} ($\dot{V}_{E\,1.5}$); tidal volume (V_T at a $\dot{V}_E$ of 30/1 min^{-1} ($V_{T\,30}$); cardiac frequency ($\dot{f}_H$) at a $\dot{V}O_2$ of 1.5/1 min^{-1} ($\dot{f}_{H\,1.5}$) and $\dot{V}O_2$ at an $\dot{f}_H$ of 195 beats min^{-1} ($\dot{V}O_{2\,195}$). All values were calculated

from the appropriate, individual regression equations. Direct measurements of maximal aerobic power output ($\dot{V}O_{2\,max}$) were not attempted. An estimate of $\dot{V}O_{2\,max}$ may, however, be made from the $\dot{V}O_{2\,195}$ though this may lead to underestimates in the younger and overestimates in the older subjects.

Thermoregulatory function tests. Heat tolerance was measured on 117 men by means of two thermoregulatory function test beds.[20] The subjects were fitted with tympanic and skin thermistors and dressed in vapor barrier suits designed for use on the heated bed. Tympanic temperature was monitored at 2-minute intervals from both ears and skin temperature recorded as the mean from eight sites. Following a short period of exposure to a neutral environment, the air temperature of the bed was lowered to 15° C for 30 minutes. The bed air temperature was then raised to 45° C until sweat onset which was detected by starch-iodine paper applied to the forehead. Finally, by adjusting the bed air temperature, the body temperature was rapidly raised to 38° C and maintained at that level for 30 minutes while sweat rate was measured. The body temperature of sweat onset and the sweat loss during the period of controlled hyperthermia were used as indices of heat acclimatization.

During the hottest climatic conditions in Sudan, in June, a further series of tests was made on a group of 16 volunteers from the Sudanese army. Prior to the thermoregulatory function tests, these men were fully acclimatized to heat by previous daily physical training and by a step-climbing procedure carried out for 2 hours a day on 5 consecutive days. During the step-climbing routine the men wore vapor-barrier suits following the method described by Marcus.[21] Immediately after the acclimatization procedure the men were tested by the same thermoregulatory function test which had been applied to the cane cutters.

Field Studies on Productivity

Measurements were made of the individual productive output of 129 cane cutters in terms of the weight of cane cut per unit time (kg min^{-1}). Representative samples were drawn from the six groups of men previously studied in the laboratory. Productivity, which was regarded as an assessment of the individual's practical working ability and capacity was measured on each man on 2 days. The first day of the study was intended to be a normal working day indicating the cutter's habitual activity. The second day was designed to measure maximum productivity and a monetary bonus system was used as inducement.

Observers (members of the scientific team) were allotted to groups of 7 to 10 men during the productivity studies. On the first day, the time taken for each man to cut five furrows along the entire length of an "hawasha" (75 meters) was recorded, and on the second day the distance cut in 2.5 hr was measured. Following the normal work procedure, the cane cut by two men was stacked in a pile between them; cane from furrows nearest to the central pile was stacked as it was cut, that from furrows furthest from the central pile was stacked after a distance has been cut. The pairs of men were chosen at random and not selected on the basis of similar productivities. When all the cane had been cut, each stack was loaded mechanically onto a single trailer and taken to the factory for weighing. By this means the total weight of cane cut by two men was obtained. The weight cut by each man was then calculated from the weight yielded by each meter multiplied by the number of furrows cut. The amount of cane cut by those men who did not complete the full quota was then estimated by the distance cut multiplied by the yield per meter.

The oral temperature of each cutter was taken before cutting started and immediately after finishing. In addition, the energy cost (oxygen consumption) of cane cutting was measured using Kofranyi-Michaelis portable respirometers. The oxygen content of expired air samples was obtained immediately after collection by means of a portable Servomex oxygen analyser which was calibrated daily. Assuming an R.Q. of 0.85, oxygen intake was converted into energy expenditure using the formula of Weir[22] in which CO_2 concentration is not taken into account. On a further 4 days, serial observations were made in the field of heart rate, body weight changes (portable beam balance) and urine temperatures.[23]

Measurements of wet and dry bulb, globe temperature, observations of cloud cover and surface air movement were made at approximately half-hourly intervals, and 3-hourly barometric pressure readings were obtained from Khartoum meteorological observations.

TABLE 2

Correlation analysis of data from laboratory and field studies. Degrees of freedom are given in parentheses

Data on	Age	*S. mansoni*	Dependents	Seasons' experience	Productivity			KM $\dot{V}O_2$	Heart rate (field)	Lab. $\dot{V}O_{2\,195}$	% $\dot{V}O_{2\,max}$	"Efficiency"
					Normal	Bonus	Mean					
Age	1.000 (187)											
S. mansoni	0.026 (187)	1.000 (190)										
Dependents	0.486* (187)	0.054 (187)	1.000 (187)									
Seasons' experience	0.264* (184)	0.286* (184)	0.245† (184)	1.000 (184)								
Productivity (normal)	−0.100 (121)	0.070 (123)	−0.058 (121)	0.166 (119)	1.000 (123)							
Productivity (bonus)	0.050 (120)	0.047 (122)	0.127 (120)	0.201† (118)	0.486* (116)	1.000 (122)						
Mean productivity	0.020 (114)	0.108 (116)	0.081 (114)	0.256* (112)	0.830* (116)	0.892* (116)	1.000 (116)					
KM $\dot{V}O_2$	0.094 (37)	−0.146 (39)	−0.072 (37)	0.130 (36)	0.459* (38)	0.493* (36)	0.564* (36)	1.000 (39)				
Heart rate (field)	−0.026 (54)	0.082 (54)	−0.015 (54)	−0.004 (54)	0.227 (51)	0.344† (49)	0.336† (48)	0.297 (37)	1.000 (54)			
Lab. $\dot{V}O_{2\,195}$	−0.119 (104)	0.068 (105)	−0.043 (104)	0.026 (102)	0.335* (98)	0.246 (98)	0.327* (92)	0.292 (29)	−0.116 (30)	1.000 (105)		
% $\dot{V}O_{2\,max}$	0.097 (28)	−0.342 (29)	0.081 (28)	−0.167 (27)	−0.196 (28)	0.033 (27)	−0.067 (27)	0.664* (29)	0.272 (26)	−0.509* (29)	1.000 (29)	
"Efficiency"	−0.190 (34)	0.278 (36)	−0.116 (34)	0.208 (33)	0.324† (36)	0.366† (36)	0.403† (34)	−0.513* (36)	0.058 (32)	0.249 (27)	−0.670* (30)	1.000 (36)

* Significant >1%, <2% level.
† Significant at or near 5% level.

TABLE 3

Physical characteristics. Age, weight (Wt), height (Ht), sum of four skinfold thicknesses (Σsf), lean body mass (LBM) calculated from skinfold thickness after the method of Durnin and Rahaman,[17] leg muscle (plus bone) volume (LV) calculated from anthropometric measurements after the method of Jones and Pearson,[18] and hemoglobin concentration. Means (± S.D.)

Group	N	Age (yr)	Wt (kg)	Ht (cm)	Σsf (mm)	LBM (kg)	LV (l)	Hb* (g/100 ml)
A	27	20.6 ± 1.7	59.2 ± 6.7	173.8 ± 6.1	22.7 ± 4.8	53.0 ± 5.7	12.6 ± 1.7	12.4 ± 1.1
B	41	21.2 ± 2.3	56.0 ± 7.0	172.3 ± 6.8	20.6 ± 3.0	50.9 ± 5.9	12.0 ± 1.9	11.5 ± 1.2
C	31	21.0 ± 2.5	57.8 ± 6.8	172.0 ± 7.3	21.8 ± 4.2	52.1 ± 6.1	12.0 ± 1.4	11.7 ± 1.4
D	26	31.9 ± 7.9	58.0 ± 7.1	172.8 ± 7.5	21.1 ± 4.3	52.5 ± 6.2	11.9 ± 1.6	12.8 ± 1.6
E	43	30.8 ± 5.5	61.1 ± 7.4	174.2 ± 6.2	23.2 ± 6.1	54.7 ± 5.4	12.4 ± 1.6	12.2 ± 1.4
F	26	32.8 ± 4.7	59.2 ± 6.1	174.8 ± 7.6	21.2 ± 3.4	53.6 ± 5.3	12.2 ± 1.7	11.9 ± 1.4

* Hemoglobin levels: A v B ($P < 0.01$); A v C ($P < 0.05$); D v E (N.S.); D v F ($P < 0.02$); B v C (N.S.); E v F (N.S.); (A + D) v (B + E) ($P < 0.002$); (A + D) v (C + F) ($P < 0.002$); (B + E) v (C + F) (N.S.).

Statistical Analysis

In the statistical treatment of the results, the six groups of cane cutters were compared for each variable by a one-way analysis of variance. A correlation analysis was carried out on data from all the subjects involved in the laboratory and field tests (Table 2).

RESULTS

Sociological Data

Of the 194 cane cutters examined in the laboratory tests, 87% originated from Kordofan and Darfur provinces which extend more than 1,000 km to the west of Guneid (Blue Nile Province). In the western provinces the level of endemicity of *S. mansoni* and malaria was much lower than in the irrigated Gezira area. Almost all of the men had migrated in November or December 1973 from townships linked by rail to the Gezira. Most worked in the cane fields for 5 or 6 months in the year and were agricultural workers (76%), herdsmen, or casual laborers by occupation. Five percent of the men were Christian, the remainder were Muslim. This seasonal migratory work force was housed in the mud-hut villages on the plantation, 136 of the subjects in one village and 58 in another. Some men brought their families to the villages during the cane-cutting season. Although the women did not actually come to the cane fields, a number of cutters were frequently assisted by relatives and friends. It was partly due to this fact that the employers records of seasonal earnings could not be used to assess individual productivity. Eighty cane cutters were celibate and 114 married, of whom only 14 had two or more wives and 75 had one or more children. A larger proportion (36%) of the younger age group (16–24 years) had no dependents as compared with the older workers (5%), and 62% of the older group had three or more dependents as compared with 28% for the younger men. Seventy-seven percent of the men professed to be nonsmokers; pipe and cigarette smoking habits of the remainder were distributed uniformly among the six groups A to F.

The number of seasons' experience at cutting cane was noted in each case and this ranged from first season (35% of the younger group and 26% of the older group) to five or more seasons (16% of the younger group and 32% of the older group). The average number of seasons' experience for the groups was 1.4 ± 1.46 S.D. (A + D), 3.5 ± 2.39 (B + E), and 3.57 ± 2.47 (C + F). Factors which appeared most likely to be related to individual productivity were the number of seasons' experience (habitual skill) and the number of dependents (motivation). These factors have been included in the correlation analysis (Table 2).

Physical Characteristics and Hemoglobin Concentration

The group distribution, age, hemoglobin concentration and physical characteristics are given in Table 3. There were no statistically significant differences between the groups in anthropometric measurements. Hemoglobin levels were, however, significantly different. In both the younger and older groups the noninfected men had higher mean

TABLE 4

Submaximal responses to exercise, oxygen intake ($\dot{V}O_2$) at a work output of 900 kpm min^{-1} ($\dot{V}O_{2\,900}$); ventilation at a $\dot{V}CO_2$ of 1.5 l min^{-1}, tidal volume at a $\dot{V}_E$ of 30 l min^{-1} ($V_{T\,30}$), cardiac frequency at a $\dot{V}O_2$ of 1.5 l min^{-1} ($\dot{f}_{H\,1.5}$) and $\dot{V}O_2$ at an $\dot{f}_H$ of 195 beats min^{-1} ($VO_{2\,195}$) in absolute (l min^{-1}) and relative (ml/kg body weight)$^{-1}$ min^{-1}; ml kg (lean body mass)$^{-1}$ min^{-1} and ml l (leg volume)$^{-1}$ min^{-1} terms

						$\dot{V}O_{2\,195}$			
Group	N	$\dot{V}O_{2\,900}$ (l min^{-1})	$\dot{V}_{E\,1.5}$ (l min^{-1})	$V_{T\,30}$ (l)	$\dot{f}_{H\,1.5}$ (beats min^{-1})	Abs. (l min^{-1})	Wt (ml kg^{-1} min^{-1})	LBM (ml kg^{-1} min^{-1})	LV (ml l^{-1} min^{-1})
A	28	2.23 ± 0.16	45.1 ± 8.0	1.21 ± 0.23	123 ± 16	2.88 ± 0.43	48.3 ± 5.7	53.9 ± 6.2	230 ± 40
B	40	2.16 ± 0.22	47.3 ± 11.1	1.15 ± 0.18	131 ± 14	2.84 ± 0.57	50.7 ± 7.1	55.7 ± 7.7	240 ± 40
C	31	2.26 ± 0.19	48.7 ± 10.4	1.11 ± 0.24	130 ± 18	2.83 ± 0.32	49.8 ± 5.4	55.1 ± 6.1	240 ± 30
D	27	2.13 ± 0.19	47.1 ± 7.8	1.27 ± 0.32	128 ± 19	2.67 ± 0.42	47.3 ± 6.4	52.0 ± 6.8	230 ± 40
E	41	2.20 ± 0.14	46.5 ± 9.3	1.24 ± 0.24	123 ± 17	2.80 ± 0.49	46.4 ± 7.6	51.6 ± 8.0	230 ± 40
F	27	2.21 ± 0.16	47.9 ± 9.5	1.20 ± 0.26	128 ± 23	2.97 ± 0.70	49.3 ± 8.7	54.4 ± 9.6	240 ± 40

hemoglobin concentrations than the respective infected groups. There was a highly significant difference ($P < 0.002$) between the noninfected group (A + D) and the infected groups (B + E) or (C + F).

Physiological Responses to Exercise

The results are summarized in Table 4. The responses to exercise were similar in the six groups measured and are in accord with previous findings for African subjects.[19] The mean $\dot{V}O_{2\,900}$, $\dot{V}_{E\,1.5}$, $V_{T\,30}$, $\dot{f}_{H1.5}$ and $\dot{V}O_{2\,195}$ in both absolute terms and relative to body weight, lean body mass and leg volume are the same and independent of age and degree of schistosomiasis infection. The correlation between $\dot{V}O_{2\,195}$ measured in the laboratory and other variables recorded during the study is given in Table 2. Thus, the data give no support to the view that *S. mansoni* impairs physiological responses to work as measured in adult males on a stationary bicycle ergometer.

Thermal Tolerance

The results of thermoregulatory function tests are given in Table 5. No significant differences were found between the six groups in physique and body composition, heart rate, core temperature and skin temperature measured at neutral temperature, or in sweat onset temperature and sweat output during controlled hyperthermia. These results suggest that there were no differences between the groups in the degree of heat stress to which they had been habitually exposed. In fact, the cane cutters produced considerably less sweat during controlled hyperthermia than the group of soldiers (Group G) who were fully heat acclimatized (Table 5). The cane cutters may be regarded as having little natural acclimatization to heat in terms of their sweating capacity.

Productivity Studies

The mean daily weight of cane cut by each man and mean productivity (kg min^{-1}) during the two normal working days and 2 days with bonuses are shown in Table 6. The mean productivity by the 129 subjects during the four field studies was 15.0 kg min^{-1} (range 6.9 to 26.5 kg min^{-1}). There was no significant difference between the productivity on the 2 normal as compared with the 2 bonus days. A good correlation was, however, found between each individual's productivity on the 2 days each cutter was observed ($P < 0.001$); the high producers were high on both days, the low producers low on both days.

In Table 7 the mean productivity of each of three groups (A + D), (B + E) and (C + F) considered in relation to the degree of infection but not age, are recorded for the 4 study days. It was found that men in group (A + D) were generally the lowest producers, group (B + E) the highest and group (C + F) intermediate. There was a statistically significant difference between groups (B + E) and (A + D) on the 1st day of the study ($P < 0.05$) and mean productivity for the 4 days of the study ($P < 0.05$). Other differences in productivity between the

TABLE 5

Physical characteristics, heart rate ($\dot{f}_H$), core temperature (T_c), skin temperature (T_s) and sweat loss at neutral temperature (N.T.), sweat onset (S.O.) and controlled hyperthermia (C.H.), during thermoregulatory function tests in cane cutters (Groups A to F) and heat-acclimatized soldiers (Group G). Means ± S.D.

Group	N	Age (yr)	Ht (cm)	Wt (kg)	S.A. (Dubois) (m^2)	$\dot{f}_H$ (beats/min)		T_c (°C)			T_s (°C)			30 min sweat loss (ml) C.H. (95% confidence interval)
						N.T.	C.H.	N.T.	S.O.	C.H.	N.T.	S.O.	C.H.	
A	19	21.1 ± 1.7	172.5 ± 5.2	58.2 ± 5.9	1.69 ± 0.10	66 ± 5	99 ± 3	37.06 ± 0.09	36.91 ± 0.10	38.04 ± 0.03	33.62 ± 0.45	36.35 ± 0.25	37.43 ± 0.09	127.0 (96.2–167.6)
B	19	20.8 ± 2.4	172.0 ± 7.0	56.1 ± 7.1	1.66 ± 0.13	68 ± 3	106 ± 3	37.00 ± 0.09	36.88 ± 0.07	38.06 ± 0.02	33.13 ± 0.34	35.86 ± 0.28	37.41 ± 0.08	150.0 (117.8–191.0)
C	18	21.0 ± 2.7	173.6 ± 6.0	58.3 ± 5.7	1.70 ± 0.09	67 ± 3	102 ± 3	37.09 ± 0.08	36.92 ± 0.08	38.07 ± 0.02	33.20 ± 0.52	36.36 ± 0.73	37.37 ± 0.06	115.2 (84.9–156.4)
D	17	32.2 ± 8.6	172.5 ± 8.8	57.4 ± 7.4	1.68 ± 0.14	71 ± 4	103 ± 3	36.99 ± 0.14	36.86 ± 0.10	38.05 ± 0.01	33.18 ± 0.48	36.07 ± 0.23	37.39 ± 0.09	164.9 (126.4–215.1)
E	25	31.8 ± 6.2	174.6 ± 6.1	61.5 ± 7.0	1.74 ± 0.12	69 ± 3	104 ± 4	37.01 ± 0.09	36.90 ± 0.10	38.05 ± 0.01	33.35 ± 0.36	36.17 ± 0.27	37.41 ± 0.09	159.4 (122.2–207.9)
F	19	32.6 ± 5.1	175.3 ± 8.0	58.7 ± 6.4	1.72 ± 0.13	71 ± 4	100 ± 3	37.08 ± 0.13	37.08 ± 0.12	38.04 ± 0.02	33.98 ± 0.38	35.68 ± 0.36	37.70 ± 0.06	152.9 (111.2–210.3)
G (Acclim.)	16	26.0 ± 3.6	173.8 ± 8.8	60.0 ± 6.7	1.72 ± 0.13	66 ± 8	98 ± 9	36.78 ± 0.22	36.69 ± 0.23	38.01 ± 0.06	34.28 ± 0.86	35.42 ± 0.49	37.59 ± 0.24	420.8 (367.2–494.6)

TABLE 6

Mean daily yield and productivity of cane cutters during field studies at Guneid

Day	No. of men	Daily yield per man (kg) Mean	(Range)	Productivity per man (kg/min) (mean ± S.D.)
1. Normal	77	2,173	(1,115–3,210)	15.04 ± 3.47
2. Bonus	76	2,729	(1,800–4,190)	16.07 ± 4.47
3. Normal	52	2,403	(1,715–3,275)	14.57 ± 2.98
4. Bonus	51	2,105	(1,475–3,000)	14.12 ± 3.07

groups were not significant. A further analysis was made using the data from only those men who had 2 or 3 seasons' experience at cane cutting. Again there was no statistically significant difference between the groups.

Oxygen consumption during cane cutting and mean cardiac frequency calculated from half-hourly recordings are given in Table 8. Oxygen intake and cardiac frequency measured during respirometry measurements were significantly correlated ($P < 0.05$). The highest oxygen intake during cutting was observed in group (B + E) which was significantly higher ($P < 0.005$) than group (C + F) or ($P < 0.05$) group (A + D). There were no statistically significant differences in mean cardiac frequency between the groups during cutting.

Correlation Analysis

A correlation matrix of the data from laboratory and field studies is given in Table 2. For the purpose of this analysis the level of *S. mansoni* infection was graded in terms of the three categories already defined. The number of seasons' cutting experience was found to be significantly correlated with age, the degree of infection and mean productivity.

The mean productivity (mean of normal and bonus days' productivity) was significantly correlated with the individual's estimated maximum aerobic capacity determined in the laboratory (Lab $\dot{V}O_{2\,195}$) and with the mean oxygen uptake measured in the field (KM $\dot{V}O_2$). When oxygen uptake as measured in the field studies was compared with the laboratory estimated $\dot{V}O_{2\,max}$

$$\left(\text{relative work rate} = \%\ \dot{V}O_{2\,max} = \frac{\text{KM } \dot{V}O_2}{\text{Lab } \dot{V}O_{2\,195}} \times 100\right)$$

it was shown that the cutters worked at about 57% of their $\dot{V}O_{2\,max}$ (range 34 to 76%). If the average relative work rates of the three groups are considered, 14 of the uninfected group (A + D) worked at oxygen intakes corresponding to 61%, 10 of the group (B + E) at 58%, and 7 of the group (C + F) at 50%. These differences were not, however, statistically significant.

TABLE 7

Mean productivity per man analyzed for noninfected and infected groups (kg/min) (± S.D.)

Day	Group A + D	Group B + E	Group C + F
1. Normal	13.93 ± 3.10* (n = 28)	15.95 ± 3.75* (n = 27)	15.30 ± 3.30 (n = 20)
2. Bonus	15.06 ± 4.43 (n = 28)	16.53 ± 3.80 (n = 28)	16.86 ± 5.33 (n = 19)
3. Normal	14.46 ± 2.93 (n = 11)	15.06 ± 2.74 (n = 20)	14.16 ± 3.29 (n = 21)
4. Bonus	13.01 ± 3.75 (n = 10)	14.33 ± 3.04 (n = 20)	14.44 ± 2.70 (n = 21)
Mean for 4 days	14.08 ± 3.03* (n = 39)	15.57 ± 3.36* (n = 47)	14.76 ± 3.33 (n = 41)

* Difference between Group (A + D) and Group (B + E) significant at 5% level.

An attempt was made to determine the "efficiency" of each worker by comparing the mean productivity with oxygen intake during cutting

$$\left(\text{where "efficiency"} = \frac{\text{mean productivity}}{\text{KM } \dot{V}O_2}\right).$$

This is a very crude index, not least because it assumes that the relationship between productivity and work rate is consistent in any one individual from day to day. We have only slender evidence that this was in fact so. Using this index, the uninfected group were apparently less efficient than the two infected groups which were similar, but none of the differences was significant.

DISCUSSION

In these investigations an attempt has been made to assess, under controlled conditions, the effect of *S. mansoni* on the physical working capacity of an active working population. The study has highlighted many of the difficulties involved in measurements of this kind, but has failed to find any clear evidence that *S. mansoni* affects standard parameters of physiological function or productive output in the normal working

TABLE 8

Mean oxygen consumption and heart rate in noninfected and infected groups during cane cutting (± S.D.)

	Group			
	(A + D)	(B + E)	(C + F)	Mean
Oxygen consumption* (l/min)	1.62 ± 0.32 (n = 18)	1.87 ± 0.35 (n = 13)	1.45 ± 0.25 (n = 10)	1.67 ± 0.34 (n = 41)
Heart rate† (beats/min)	132 ± 16 (n = 24)	139 ± 15 (n = 17)	134 ± 9 (n = 15)	135 ± 14 (n = 56)

*(A + D v (B + E) ($P < 0.05$); (A + D) v (C + F) ($P < 0.005$).
†Derived from the average of 3 to 8 heart rates observed at half-hourly intervals on each subject.

situation. These findings confirm those of a number of other investigators,[6, 7, 14, 24] and raise the question as to why the discrepancy exists between the widely held view that schistosomiasis seriously prejudices working capacity and the lack of substantive experimental data to support it. The recent observations of Omer and El Din Ahmed[8] on young adult male nurses infected with *S. mansoni* suggest that there may be some physiological parameters which are affected. Forced expiratory flow ($FEF_{200-1,200}$) was found to be reduced (but not five other standard tests of lung function including FVC and FEV) as was a "physical condition index" based on post-exercise cardiac frequency and cardiac frequency (but not oxygen consumption) during exercise. It is not entirely consistent that only some of the physiological indices appear to be altered by the disease, but it is perhaps more significant that after treatment with niridazole these authors found less fatigueability and an improvement in the $FEF_{200-1,200}$ and "physical condition index." The patients of Omer and El Din Ahmed do not appear to have been more severely infected than ours, and in fact had no hepatic or splenic enlargement as did our groups C and F. One reason for the difference in findings may lie in the type of exercise test performed. The standard bicycle ergometer test was undoubtedly a novel form of exercise for the cane cutters as compared with the stepping test used for the male nurses. Although a period of habituation preceded the ergometer test, this may not have been long enough to ensure a uniform starting point for all the subjects. Nevertheless, this objection would have applied to all groups since the subjects were selected for testing in a random order. General fitness and muscular power was probably higher in the cane cutters than the male nurses, and any adaptive physiological mechanism which allowed for improvement in physical work performance in the presence of *S. mansoni* infection would be more likely to obscure differences between infected and noninfected groups in the cane cutters.

One clear difference between the infected and noninfected cane cutters appears to be in hemoglobin concentrations which overall were in the lower quartile of the normal range but not below 10 g/100 ml. The anemia of schistosomiasis is reported to be of the hemolytic type initiated by an immune process.[26] Such a process could account for the slight but significantly greater degree of anemia in the intestinal group without splenomegaly as compared with the noninfected group. In African males, iron-deficiency anemia (Hb 6.7 to 9.2 g/100 ml) has been shown to reduce maximum aerobic power and increase cardiac frequency during exercise on a bicycle ergometer.[27] The differences between mean hemoglobin concentrations in the groups of cane cutters were at most 1.3 g/100 ml and hardly likely to have had any significant effect on the physiological variables measured during exercise.

Paradoxically, it was found in the field productivity studies that group (B + E) had the highest average productivity, 10% higher than group (A + D) which was significant, and 5% higher than group (C + F). However, when the daily productivity of each group was examined, on only one occasion was there a significant difference between groups, so that there is really no consistent difference in group productivities. The most likely explanation for the fact that the noninfected group had the lowest productivity is that many of these men were in their first season of cane cutting and therefore less skilled than the more experienced men. There was no significant correlation between seasons' experience and objective physiological

measurements that suggested a difference in physical fitness. The averall range of individual productivities observed is so wide (375% between the highest and lowest) that despite a 25% coefficient of variability about the mean group productivity it seems certain that if *S. mansoni* infection had any deleterious effect on physical performance in any of the groups the productivity studies would have revealed it. All the groups had similar average heart rates during cutting which suggests that they were all exerting themselves to a similar degree.

In the field studies the average metabolic rates of groups (B + E) and (C + F) during cane cutting were significantly different. This suggests that respirometry during occupational activity could be a useful method of detecting differences in physical working capability between groups of subjects. An activity such as cane cutting imposes considerable demands on the physical working capacity, and respirometry is sufficiently sensitive to detect relatively small difference in metabolic rate. In a disease such as schistosomiasis it may be the patient's ability to sustain a high work output that is impaired rather than his absolute maximal working capacity.

The cane cutters at Guneid were all volunteers and in spite of schistosomiasis were fit enough to perform hard physical work with high average oxygen consumptions. In this respect, therefore, they were highly selected and would not necessarily provide a true reflection of the effect of schistosomiasis on the working population as a whole. Those, for example, who were more seriously debilitated may have been absent from work and automatically excluded from this study. In investigations of earnings on sugar estate workers in Tanzania,[25] noninfected workers were found to earn at least 11% more in bonuses than those infected with *S. mansoni*. But the significant decrease in productivity in those infected, estimated to be of the order of 5%, was associated with a higher incidence of absenteeism. The cane cutters in our investigation, in effect, were a fit, migrant population, many of whom were suffering only from the acute effects of *S. mansoni* infection. A better index of the human and economic impact of the disease is more likely to be obtained in a static local population where the chronic ill effects of the disease have become more apparent.

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Temperature Regulation during Exercise in Water and Air

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Abstract

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Four healthy subjects were studied during exercise in water, using a swimming flume, and in air, on a stationary bicycle ergometer at mean skin temperatures of 30 and 33°C, respectively. Measurements included rectal (T_{re}), esophageal (T_{es}), and mean skin ($\overline{T}_s$) temperatures, metabolic energy liberation (M) and total heat production (H), maximal aerobic power output ($\dot{V}_{O_2\,max}$), cardiac frequency and calculated peripheral tissue heat conductance (K). The results showed that for a given M and $\overline{T}_s$, T_{es} and T_{re} were about 0.4°C lower and the K values were consistently higher in swimming than in bicycling. The intersubject variability in T_{es} and T_{re} was reduced by considering relative (expressed as $\% \dot{V}_{O_2\,max}$) rather than absolute work load, but the differences in the body temperatures between the two types of exercise remained. It was concluded that during exercise in water where the capacity for heat dissipation is increased, the body core temperature (T_c) is maintained at a lower level due to the higher forced convective and conductive heat transfer from the skin in water. This reduces the heat storage at the beginning of exercise compared with conditions in air. The lower $T_c - \overline{T}_s$ gradient for a given H in swimming, which results in higher K values implies a greater skin circulation than during cycling in air.

Key words: Esophageal temperature, environment, work, temperature regulation, skin temperature, sweat rate, heat production, heat dissipation

There have been several studies of temperature regulation during exercise (see B. Nielsen 1969 for general review). From these investigations it has emerged (M. Nielsen 1938) that the rise in core temperature (T_c) is proportional to the work load and metabolic energy liberation (M) independent of environmental conditions over a wide range. However, though the T_c/M relationship may hold for a given individual, it is clear from the results of Åstrand (1960) and Saltin and Hermansen (1966) that when groups of subjects are considered, the rise in T_c is related to the relative (expressed as a % of the maximal aerobic power output) rather than to the absolute metabolic energy liberation. These findings suggest that the rise in T_c during exercise may not only be governed by M but also by the available circulatory capacity for heat transfer from the core to the periphery. The difficulty of studying this problem further is that the heat flow across and from the skin is affected by

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$\bar{T}_s$ and the thermal properties of the environment. In the present experiments we have investigated the responses of 4 healthy subjects to exercise at a given M and constant $\bar{T}_s$ in air and water; two environments which differ greatly in their heat transfer capacity.

Materials and Methods

The swimming expts. took place in a swimming flume (Åstrand and Englesson 1972), where the speed and temperature of the water could be regulated between 0 and 2 m/s and between 10° and 40°C respectively. The subjects swam in water at 2 temperatures of 30 or 33°C, and at a constant speed which they could maintain for 1 h using the breast stroke. Core temperatures were measured with thermistor probes placed at 15 cm depth in the rectum (T_{re}) and just above the diaphragm in the esophagus (T_{es}). The placing of the esophagus thermistor was controlled by X-ray photography. The signals were continuously transmitted through a radio transmitter system (Danica electronic) suspended above the swimming subject, to a receiver connected to a Servogor recorder, so that no high voltage equipment was close to the subject in the water. The ECG was similarly transmitted. The accuracy of the temperature measurements were ± 0.1°C. The thermometers were calibrated in well stirred thermos flasks after each expt.

The $\dot{V}_{O_2}$ was measured with the Douglas bag method approximately every 15 min during the expt. The subject breathed in a special valve supplied with a snorkel. Expired air was collected in 150 l plastic bags placed on a large wire net above the swimmer. The air volumes were measured in a Collin's spirometer and analysed for O_2 and CO_2 on a Servomec (OA 180) paramagnetic O_2 analyser and a Beckman LB 1 infrared CO_2 analyser. The reference gases were checked with the Scholander analyser technique (Scholander 1949).

The bicycle expts. took place in a climatic chamber at air temperatures which resulted in average skin temperatures ($\bar{T}_s$) of respectively 30° and 33° as in the water expts. Air temperature and velocity in the room were sometimes changed during the expt. in order to obtain the required $\bar{T}_s$.

The exercise was performed on a Krogh bicycle ergometer suspended in the Krogh balance as described previously (Nielsen and Nielsen 1965). The subjects worked at one or two loads which gave the same $\dot{V}_{O_2}$ (measured every 15 min) as the swimming expts. (*cf.* Table II). Core temperatures (T_{es}) and (T_{re}) were measured with thermocouples as described earlier (Nielsen and Nielsen 1962) and recorded on a Kipp Micrograph BD I.

The temperature of 15 skin areas was measured every 5–10 min with a thermocouple (Nielsen and Nielsen 1965). An average skin temperature ($\bar{T}_s$) was then calculated by weighting the 15 measurements in relation to the size of the corresponding skin area (Hardy and DuBois 1938). Evaporative sweat loss was calculated from weight changes recorded continuously on the Krogh balance (Krogh and Trolle 1936), corrected for respiratory weight loss due to evaporation and gas exchange. From the raw data the following calculations were made:

Rate of metabolic energy production in watts: $M = \dot{V}_{O_2} \times 341$ W

Rate of heat production in watts: $H = M - W$, where

W is the external work. The additional work performed to overcome friction in the chain transmission was not measured, and for our purpose negligible. During swimming the external work was ignored since the net efficiency is very low 1–5 % (Karpovich and Pestrecov 1939, Holmér 1974).

Rate of heat storage in watts:

$S = (T_c \times 0.65 + \bar{T}_s\ 0.35) \times h^{-1} \times$ body weight $\times 0.97$

0.97 = body specific heat ($W \times h \times kg^{-1} \times °C^{-1}$)

T_c = mean core temp. $= \dfrac{T_{es} + T_{re}}{2}$

In the swimming experiment $\bar{T}_s$ was considered constant and equal to water temperature.

Conductance $K = \dfrac{H - S - E_L}{(T_c - \bar{T}_s)\ A_D}$ $W \times M^{-2} \times °C^{-1}$

where E_L = evaporate heat loss from lungs and A_D = body surface. Conductance was calculated from values measured between 45 and 60 min. The subjects were young students with average to excellent swimming abilities. Their physical characteristics are presented in Table I.

TABLE I. Physical characteristics: Age, height (ht), weight (wt), Du Bois (surface) area (A_D), the average skinfold thickness from 21 sites (SF) and maximal aerobic power output ($\dot{V}_{O_2}max$).

Subject	Sex	Age (yr)	Ht (cm)	Wt (kg)	A_D m^2	SF mm	$\dot{V}_{O_2}max$ l × min^{-1}	
							Bicycling	Swimming
VBP	♀	32	174	54	1.64	8.5	2.74	—
MNO	♂	25	186	80	2.04	8.3	5.19	4.86
HL	♂	25	189	85	2.13	9.4	4.17	—
LA	♂	23	175	75	1.89	8.8	4.14	3.77

Results

The basic data are summarised in Table II. A comparison of the metabolic rate, heart rate, temperature responses and peripheral tissue conductances during swimming and bicycling are presented in Fig. 1. The subjects exercised with the same metabolic rate (M) (Fig. 1) and $\overline{T}_s$ (Table II) in the two experimental situations but the heart rates were higher in bicycling than in swimming in all the expts. at $\overline{T}_s = 33°$, and in 2 of the 6 expts. at $\overline{T}_s = 30°$. The T_{es} and T_{re} were on average 0.4°C lower in swimming than in bicycling, and this lower body temperature was reflected in the high K values for a given M in swimming at 30 and 33°C (Fig. 2).

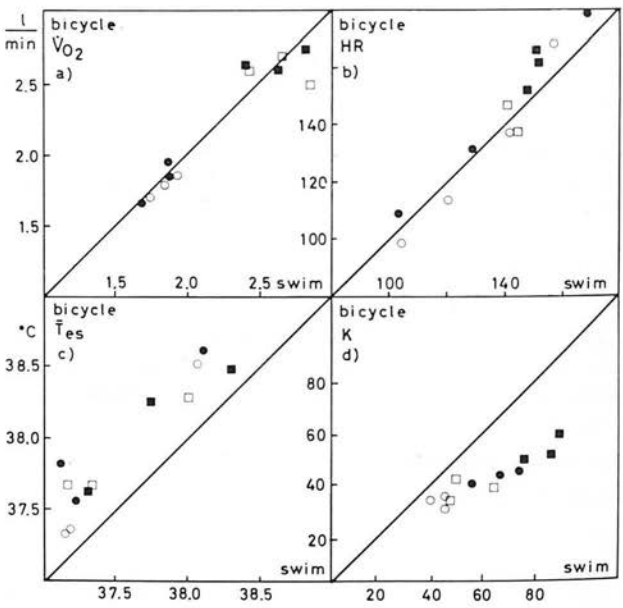

Fig. 1. A comparison of (a) $\dot{V}_{O_2}$, (b) cardiac frequency, (c) esophageal temperature and (d) peripheral tissue heat conductance (K) in bicycling with swimming. 2 workloads and 2 skin temperatures. 4 subjects.

	30°	33°
I $\dot{V}_{O_2} \approx 1.6$ 1/min	○	●
II $\dot{V}_{O_2} \approx 2.6$ 1/min	□	■

TABLE II. Metabolic and thermal responses to 2 levels of exercise (I and II) in water and air at mean skin temperatures ($\bar{T}_s$) of 30°C and 33°C. Oxygen intake ($\dot{V}_{O_2}$) relative work load (% $\dot{V}_{O_2max}$); heart rate (HR), metabolic (M) and total heat (H) production; peripheral tissue heat conductance (K), sweat rate (SW) and mean skin ($\bar{T}_s$) temperature during the 40–60th min of exercise and mean esophageal (T_{es}) and rectal (T_{re}) temperatures during final 10 min of work.

Work load	T_s	Exercise	Subject	$\dot{V}_{O_2}$ l·min^{-1}	$\dot{V}_{O_2max}$ %	HR beats·min^{-1}	M W	H W	K W·m^{-2}·°C^{-1}	SW g·h^{-1}	T_{es} °C	T_{re} °C	$\bar{T}_s$ °C
I	30°	Swimming	V	1.85	68	156	631	631	45.4	—	38.1	38.0	30.2
			M	1.71	33	105	583	583	39.6	—	37.2	37.4	30.3
			H	—	—	—	—	—	—	—	—	—	—
			L	1.93	47	121	658	658	44.9	—	37.2	36.8	29.7
			Average	1.83	49	127	624	624	43.3	—	37.5	37.4	30.1
		Bicycling	V	1.80	66	169	614	487	30.3	403	38.5	38.7	29.8
			M	1.74	34	99	594	474	33.6	493	37.4	37.7	31.0
			H	—	—	—	—	—	—	—	—	—	—
			L	1.87	45	114	638	521	34.6	409	37.4	37.5	29.7
			Average	1.80	48	127	615	494	32.8	435	37.8	38.0	30.2
	33°	Swimming	V	1.88	69	168	641	641	68.3	—	38.1	38.2	32.9
			M	1.68	32	103	573	573	56.3	—	37.2	37.6	32.6
			H	—	—	—	—	—	—	—	—	—	—
			L	1.87	45	129	638	638	74.9	—	37.1	37.2	32.9
			Average	1.81	49	133	617	617	66.5	—	37.5	37.7	32.8
		Bicycling	V	1.86	68	180	635	508	44.3	563	38.7	38.8	32.8
			M	1.67	32	109	570	450	40.8	512	37.6	37.8	32.7
			H	—	—	—	—	—	—	—	—	—	—
			L	1.96	47	132	668	538	44.9	1 021	37.8	37.9	32.8
			Average	1.83	49	140	624	499	43.3	699	38.0	38.2	32.8
II	30°	Swimming	V	—	—	—	—	—	—	—	—	—	—
			M	2.67	51	142	911	911	49.4	—	37.2	37.8	30.7
			H	2.43	58	140	828	828	46.9	—	38.0	38.3	30.2
			L	2.86	69	144	976	976	64.7	—	37.3	37.3	29.9
			Average	2.65	59	142	905	905	53.7	—	37.5	37.8	30.3
		Bicycling	V	—	—	—	—	—	—	—	—	—	—
			M	2.69	52	138	918	734	42.1	710	37.7	38.0	29.8
			H	2.61	63	147	890	705	33.9	628	38.3	38.4	29.8
			L	2.50	60	135	853	673	38.6	749	37.7	37.9	29.9
			Average	2.60	58	140	887	704	38.2	696	37.9	38.1	29.8
	30°	Swimming	V	—	—	—	—	—	—	—	—	—	—
			M	2.42	47	147	825	825	91.2	—	37.3	37.6	33.0
			H	2.82	68	150	962	962	76.9	—	38.3	38.4	33.0
			L	2.63	64	151	897	897	88.3	—	37.8	38.0	32.9
			Average	2.62	60	149	895	895	85.5	—	37.8	38.0	33.0
		Bicycling	V	—	—	—	—	—	—	—	—	—	—
			M	2.64	51	153	900	716	60.1	778	37.7	37.7	32.4
			H	2.75	66	167	938	748	49.8	768	38.5	38.5	32.8
			L	2.61	63	162	890	710	52.1	1 241	38.3	38.5	32.5
			Average	2.67	60	161	909	725	54.0	929	38.2	38.2	32.6

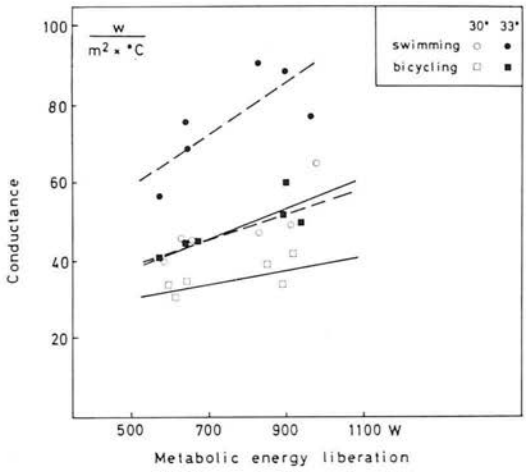

Fig. 2. Relationship between peripheral tissue and heat conductance (K) and metabolic energy liberation (M)

30° 33°
swim. ○--- ●
bicyc. □— ■ 4 subjects

The T_{es} and the T_{re} had different relationships to M and to H in swimming and bicycling. The intersubject variation of T_{es} and T_{re} was reduced by considering relative work load but the difference between T_{es} (or T_{re}) for the two forms of exercise remained (Fig. 3).

In Table III the 'forced' heat losses from the skin are presented. During swimming it is

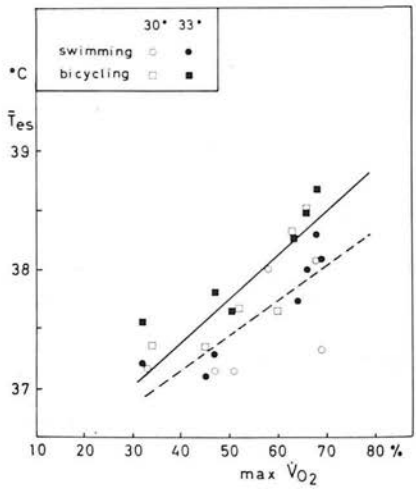

Fig. 3. Esophageal temperature (T_{es}) in relation to relative work load.

30° 33°
swim. ○--- ●
bicyc. □— ■ 4 subjects

The relative work load for swimming was calculated in terms of the $\dot{V}_{O_2}$ observed, expressed as a % of maximal aerobic power of bicycling. If the $\dot{V}_{O_2\,max}$ for swimming is used the difference between the two types of exercise widened.

TABLE III. Forced heat loss during swimming (W/m^2), calculated as minimum heat loss through skin (29 W/m^2 °C) times core to skin gradient (37°–30°C and 37°–33°). Forced heat loss during bicycling, calculated from the heat balance equation as $C+R=(H-E_{sw}-E_1-S)$. Regulatory heat loss in both types of exercise is calculated as total heat production minus the "forced heat" loss.

	30°		33°	
	H "forced"	H regulatory	H "forced"	H regulatory
Work I				
Swimming	203	137	116	221
Bicycling	95	173	−28	300
Work II				
Swimming	203	248	116	328
Bicycling	78	271	−12	371

calculated from the minimal heat conductances found for the 4 subjects in water immersion expts. previously described (Nielsen 1976), and during cycling from the heat balance equation $C+R=(H-E_{SW}-E_L-S)$. At 30°C and 33°C during work in water, the convective and conductive heat losses are on an average about 125 W m^{-2} greater than those for convection and radiation at the same $\bar{T}_s$ and M in air during cycling. Thus, the total amount of heat which requires active regulation (H_R) is at work load I 21%, respectively 26% less in swimming than in bicycling at 30° and 33°C. The corresponding figures at work load II are 9% and 12%.

Discussion

The design of our expts. was such that each subject was required to exercise at the same M and $\bar{T}_s$ in water and in air (Table II). In the swimming expts. we have assumed that $\bar{T}_s$ is equal to the water temperature. Actually $\bar{T}_s$ is somewhat higher than water temperature, depending upon exposure time, velocity of the water and water temperature (Burton and Bazett 1936, Craig and Dvorak 1968, Keatinge 1969, Boutelier 1973); but Nadel *et al.* (1974) have shown that at 33°T_{H_2O} a gradient of only 0.2°C exists between skin and water for subjects swimming at speeds between 0.5–0.95 m/s. Further, in our calculations of H we have neglected the energy spent as external work in the swimming expts. Using the methods described by di Prampero *et al.* (1974), Holmér (1974) measured mechanical efficiency for breast stroke in the swimming flume. Holmér reports values from 1.1–5.7%, the higher efficiencies being found in elite swimmers. In ignoring external work we have probably overestimated total heat production in the swimming expts. by 3–6%. These factors will affect our calculations of K and must be borne in mind when interpreting our data. For a given M and $\bar{T}_s$, T_{es} and T_{re} were about 0.4°C lower in swimming than in cycling (Fig. 1). This resulted in a lower core–skin temperature gradient and in a higher calculated peripheral conductance (K) (Fig. 1 and 2).

It is well known that the plateau core temperature in exercise can be modified by factors which influence the convective heat transfer to, and the evaporative heat loss from the skin (Nielsen 1971, 1974 a and b). During exercise in water evaporative sweat loss is suppressed

and convective heat transfer is increased (Fig. 1). It would, therefore, seem reasonable to suppose that either one or both of these factors are implicated in the lower core temperature observed during swimming.

The heat conductance and specific heat of water are about 25 and 1000 times greater, respectively, than those of air. In our climatic chamber the combined convective and radiation heat transfer coefficient (h_0) for cycling is of the order of 17 W $\times$ m^{-2} $\times$ °C^{-1} (Nielsen 1969). The heat transfer coefficients for water (h_w) at various temperatures and speeds are less well known. They have been studied in physical models (Winterspoon *et al.* 1971 and Bullard and Rapp 1970) and a value for swimming in water between 30–33°C in a flume was measured by Nadel *et al.* (1974). They found that h_w was independent of water speed and equal to 580 W $\times$ m^{-2} $\times$ °C^{-1}.

We now define as 'forced' heat loss (H_F) the heat lost to the environment by radiation, conduction and convection, *i.e.* the heat loss due solely to the difference between the temperature of the skin and the environment.

This seems justified, because $\bar{T}_s$ in air is equal in rest and work in a given environment (B. Nielsen 1969). Therefore, $C + R$ heat loss is the same in rest, where Sw and skin blood flow are at minimum values, and during work when "regulatory heat loss" i.e. sweating and skin circulation are activated. (For a detailed discussion *cf.* Nielsen p. 31, 1969.)

From the difference between h_w and h_0 it therefore becomes clear that the 'forced' heat loss in water must be greater than in air. Our calculations (Table III) show the difference to be of the order of 125 W m^{-2}. The rest of the heat ($H - H_F = H_R$) is the "regulatory" heat, which has to be dissipated by active physiological adjustments. H_R is smaller during swimming and it is, therefore, not surprising to find a reduction in T_{es} and T_{re} during exercise in water. It merely suggests that the underlying regulating mechanisms in swimming and bicycling are similar and that the rise in core temperature may be a function of the amount of heat requiring active regulation and the rate at which heat loss can be increased.

Our results with bicycling generally confirm previous findings of Saltin and Hermansen (1966), that T_{es} and T_{re} are related to the relative work load and are independent of environmental conditions (*cf.* M. Nielsen 1938). In both situations a steady state plateau can be reached in 30–40 min, however, at a low T_c in water.

Also in the swimming expts. the interindividual differences are reduced if the core temperatures are plotted against relative work load (% of $\dot{V}_{O_2\,max}$). But compared to the bicycle expts. the slope of the $T_{es}/\%\,\dot{V}_{O_2\,max}$ relationship line is reduced (not statistically sign.) (Fig. 3), probably due to the reduced H_R as discussed above.

The meaning of this relationship is not clear. Since max $\dot{V}_{O_2}$ and maximal cardiac output are closely related, it may indicate that the proportion of the cardiac output which is used in the convective heat transfer from core to skin: $H_{skin} \approx f(T_{core} - T_s) \times Q_{skin}$, is determined by the individual's cardiovascular capacity. A rise in the core temperature would reduce the skin blood flow (SBF) needed for this heat transfer, and this rise seems to be regulated in proportion to max $\dot{Q}$.

Contrary when for other reasons T_{core} during work is reduced, as in swimming, the skin blood flow must be increased. This is demonstrated by the higher values for K in swimming found in all subjects (Fig. 2).

We conclude that the rise in core temperature during exercise is set by the change of heat stores at the beginning of exercise, and is governed by the total amount of heat requiring 'active' dissipation. A rise in body temperature will promote the convective transfer of heat from the core to periphery. The plateau level of T_c and its precise relationship to SBF will be determined by the cardiovascular capacity of the individual, as indicated by his $\dot{V}_{O_2\,max}$, and the nature of the environment. In air these factors must be finely co-ordinated with the capacity and sensitivity of the sweating mechanism. In water, the heat transfer coefficients are so large that small changes in vasomotor reactions quickly will adjust heat balance at the new level with only a small increase in heat storage and T_{core}. The present expts. underline the importance of these changes but do not explain how they are finely integrated during exercise.

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Effects of β Blockade and Atropinisation on Plasma Catecholamine Concentration during Exercise

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Abstract. The changes in plasma catecholamine concentration (ΔC) following β-blockade (practolol, 15 mg) and atropinisation (Atropine, 1.8 mg) have been studied on 5 healthy male subjects during exercise on a motor driven treadmill.

The results showed that for a given $\dot{V}O_2$ and % $\dot{V}O_{2\ max}$, β blockade was without effect on ΔC (except in one athletic subject), but atropine produced a rise in ΔC. In relation to $\dot{Q}$, both drugs produced an increase in ΔC, but for a given cardiac frequency (f_H) ΔC was higher with β blockade, and lower with atropinisation than found in control experiments. The intra- and inter-subject variability of ΔC in relation to f_H was resolved by considering the change in cardiac frequency calculated from baseline value obtained during walking at 6.44 km/h on the level, and expressed as a percentage of the maximal f_H attainable for given individuals under the different drug and control conditions (% Δf_H).

It was concluded that during short term exercise, the rise of ΔC in relation to % Δf_H reflects both the myocardial sensitivity to vagal and β blockade, and the circulatory vasoconstrictor control of blood vessels which is required to sustain increases in systemic and muscle blood flow.

Key words: Practolol – Atropine – Work – $\dot{V}O_{2\ max}$ – Cardiac frequency – Catecholamines.

Introduction

In a previous paper from this laboratory [4] we have shown that during dynamic exercise above 70% of an individual's maximal aerobic power output ($\dot{V}O_{2\ max}$), plasma catecholamine concentration (C) rises rapidly. For a given type of exercise the increase in C was associated with an increase in cardiac output ($\dot{Q}$) and with an increase in the relative work load expressed in terms of % $\dot{V}O_{2\ max}$. However, when different modes of work were considered, these two variables could be replaced by cardiac frequency (f_H). The association of f_H with C accounted for ~ 80% of the

variance of the two variables and the relationship was independent of whether the subjects used their arms, legs, or arms and legs combined to perform the work, under hypoxic, normoxic, or hyperoxic conditions. From the results of the study we suggested that the rise in C and f_H in exercise was associated with (but not necessarily causally related to) circulatory stress, and may have reflected the degree of vasoconstriction in "non-active" tissues, necessary to maintain systemic blood pressure during work.

To investigate this problem further we have measured the changes in catecholamine concentration during exercise on 5 subjects following β adrenergic and parasympathetic blockade.

Material and Methods

Five healthy male volunteers were used, whose mean ($\pm$ SD) age, weight, height and $\dot{V}O_{2\ max}$ were 35.6 $\pm$ 8.1 yr; 67.6 $\pm$ 7.3 kg; 175.1 $\pm$ 5.1 cm; and 3.86 $\pm$ 0.60 l $\cdot$ min^{-1} respectively. All but one of the subjects were normally active but not in training; the exception was JB, an endurance athlete and a member of the Road Runners Club of Great Britain. He had a $\dot{V}O_{2\ max}$ of 4.8 l $\cdot$ min^{-1} (75 ml $\cdot$ kg^{-1} $\cdot$ min^{-1}).

On three separate occasions each subject was required to exercise on a motor driven treadmill set at a constant speed, and at five or more different gradients which spanned the range of each subject's exercise tolerance. Exercise tests were conducted under control conditions, and following an intravenous injection of 15 mg practolol or 1.8 mg atropine into the antecubital vein. Each work level was performed for 10 min, and in the final 3 min various physiological measurements were made; in addition 12 ml venous blood was taken through an indwelling catheter for the analysis of total catecholamine concentration.

The physiological measurements included minute ventilation and oxygen intake which were determined by an open circuit technique utilising either a Parkinson Cowan (CD_4) dry gas meter and a mixing chamber [5], or (more often), a Tissot (Collins Ltd.) Spirometer to collect expired air.

The subjects breathed through an Otis-McKerrow low resistance valve and vented expired air into the mixing chamber or spirometer for analysis of the O_2 and CO_2 concentrations, using paramagnetic (Servomex LTd.) and infra red (Beckman Ltd.) analysers, respectively. The analysers were calibrated at frequent intervals with gases whose content was previously determined by direct Lloyd-Haldane chemical analysis. The electrocardiograph and cardiac frequency were monitored from chest electrodes during the administration of the drug (30 min before exercise), and during and following each exercise period. The chest electrodes were connected to a differential amplifier, and the output was displayed on an enlarged oscilloscope screen and an instantaneous linear rate meter [14]. $P\bar{v}_{CO_2}$ was measured using a CO_2 rebreathing technique [12] in which the subject was given an appropriate volume and concentration of CO_2 in O_2 to rebreathe for 12–15 s. The end tidal PCO_2 was recorded continuously to identify a constant 6–10 s plateau in the respired CO_2 tension due to the equilibriation of the mixed venous with the alveolar and rebreathing bag CO_2 tensions prior to recirculation. If a perfect plateau was not obtained, the extrapolation procedure described by Denison et al. [6] was used. Cardiac output was calculated using the method of Godfrey [9]; no correction was applied for the difference between the alveolar plateau PCO_2 and blood arriving or leaving the lungs, as described by Jones et al. [11].

Blood catecholamine concentration was determined using the semi-automated method of McCullough [13]. The coefficient of variation of duplicate samples was $\pm$ 6.6%. The normal maximal aerobic output was determined on a separate occasion using a technique previously described [3].

Results

The basic data for the 5 subjects are summarised in Table 1.

In Figure 1 the changes in ΔC are plotted against $\dot{V}O_2$, % $\dot{V}O_{2\ max}$, f_H and Δf_H for 1 subject, and are representative of the group excluding the athlete. Compared to

Table 1. Effects of atropine and β blockade on oxygen intake ($\dot{V}O_2$) relative work load (% $\dot{V}O_{2\,max}$), cardiac frequency (f_H Δf_H and $\Delta\% f_H$ – see text), cardiac output ($\dot{Q}$) and plasma catecholamine concentration (C and ΔC) during exercise

Subject	Gradient %	$\dot{V}O_2$ l · min^{-1}	% $\dot{V}O_{2\,max}$ %	f_H beats · min^{-1}	$\dot{Q}$ l · min^{-1}	C µg/100 ml^{-1}	ΔC	Δf_H beats · min^{-1}	$\Delta\% f_H$ %
Control experiments									
EZF	0	1.36	33	97	13.2	0.84	0	0	0
	6	1.74	42	125	12.7	0.90	0.06	28	27
	9	2.17	53	144	15.0	1.50	0.66	47	46
	12	2.63	64	165	16.6	2.22	1.38	68	66
	15	2.32	81	191	17.6	5.22	4.38	94	91
EZF	0	1.15	28	92	9.5	0.72	0	0	0
	3	1.49	36	95	11.5	–	–	3	3
	6	1.80	44	117	12.5	0.96	0.24	15	14
	9	1.96	48	130	13.2	1.38	0.66	28	26
	12	2.47	60	168	14.9	1.68	0.96	76	61
	15	2.96	72	173	16.9	2.96	2.24	81	75
	18	3.37	82	187	18.2	5.94	5.22	95	88
	20	3.66	89	200	–	7.32	6.60	108	100
CTMD	0	1.26	36	115	8.7	1.00	0	0	0
	3	1.50	42	128	10.5	1.08	0.08	13	19
	6	1.76	50	145	11.6	1.98	0.98	30	45
	9	2.07	60	159	13.1	2.76	1.76	44	65
	12	2.47	71	173	15.0	3.30	2.30	58	87
	15	2.89	83	182	18.1	6.60	5.60	67	100
JF	0	1.24	40	95	8.9	0.60	0	0	0
	3	1.39	45	108	9.7	0.72	0.12	13	14
	6	1.79	58	127	11.6	0.96	0.36	32	36
	9	2.22	72	155	14.5	–	–	–	–
	12	2.64	85	178	17.7	4.80	4.20	83	92
	15	–	–	185	–	–	–	–	–
RR	0	1.13	29	105	11.9	0.42	0	0	0
	3	1.70	44	127	13.5	0.66	0.24	22	24
	6	2.07	53	146	16.7	1.32	0.90	41	46
	9	2.28	58	163	15.5	–	–	58	64
	12	2.79	71	179	17.7	2.40	2.02	74	82
	15	–	–	186	–	–	–	81	90
JB	0	2.53	53	125	–	0.63	0	0	
	3	2.92	60	142	–	1.13	0.51	17	28
	6	3.59	74	155	–	1.56	0.39	30	50
	9	4.27	89	167	–	2.52	1.89	42	69
	12	4.83	100	186	–	7.44	6.81	61	100

control values it will be seen that for a given $\dot{V}O_2$, ΔC is raised following atropinisation but is unaffected by β blockade (Fig. 1a). Assuming that $\dot{V}O_{2\,max}$ is unchanged by the administration of Atropine and practolol (see [8]), these changes will be the same for relative work load (Fig. 1b). When cardiac frequency is considered, the two drugs are shown to have separate effects; for a given f_H practolol is associated with a higher, and atropine with a lower ΔC (Fig. 1c).

Table 1 (continued)

Subject	Gradient %	$\dot{V}O_2$ l · min^{-1}	% $\dot{V}O_{2\,max}$ %	f_H beats · min^{-1}	$\dot{Q}$ l · min^{-1}	C µg/100 ml^{-1}	ΔC	Δf_H beats · min^{-1}	$\Delta\%f_H$ %
Atropine									
EZF	0	1.27	31	125	9.7	0.72	0	0	0
	3	1.42	35	133	10.2	–	–	8	11
	6	1.78	43	149	12.5	–	–	24	32
	9	2.13	52	165	15.3	1.44	0.72	40	53
	12	2.57	63	180	16.4	–	–	55	73
	15	3.03	74	190	17.1	4.44	3.72	65	87
	18	3.46	84	197	–	6.20	5.48	72	96
EZF	0	1.24	30	133	10.7	0.54	0	0	0
	3	1.60	39	145	10.2	0.78	0.24	12	18
	6	1.92	47	156	14.9	0.96	0.92	23	34
	9	2.30	56	171	17.8	1.86	1.32	38	57
	12	2.80	68	182	21.1	2.46	1.92	49	73
	15	3.15	77	193	19.9	2.76	2.22	60	89
CTMD	0	1.21	36	115	8.7	1.00	0	0	0
	3	1.40	42	128	10.5	1.08	0.08	13	19
	6	1.70	50	145	11.6	1.98	0.98	30	45
	9	1.99	60	159	13.1	2.76	1.76	44	65
	12	2.37	71	173	15.0	3.30	2.30	58	87
	15	2.73	83	182	18.1	6.60	5.60	67	100
JF	0	1.12	36	118	8.9	0.60	0	0	0
	3	1.38	41	128	10.0	0.96	0.36	10	15
	6	1.80	58	145	12.8	1.32	0.72	27	40
	9	2.13	69	159	14.8	1.80	1.20	41	61
	12	2.69	86	176	18.7	2.82	2.22	58	87
RR	0	1.17	30	111	8.8	1.02	0	0	0
	3	1.40	36	137	11.6	1.56	0.36	26	31
	6	1.71	44	156	12.1	2.20	1.18	45	54
	9	2.06	53	168	12.9	–	–	57	68
	12	2.41	62	183	14.5	4.03	3.03	72	85
JB	0	2.42	51	140	15.3	0.72	0	0	0
	3	2.89	60	150	18.3	1.02	0.30	10	22
	6	3.44	72	165	21.6	3.12	2.40	25	54
	9	4.05	85	177	24.0	4.50	3.78	37	80
	12	–	–	183	–	19.20	18.48	43	93

However, if the change in f_H from the value found during walking at 0% gradient is calculated, then for a given Δf_H atropinisation gives a higher ΔC, while β blockade gives values which are similar to control values (Fig. 1d). The athlete (JB) showed similar relations of ΔC to the physiological variables described for the more "sedentary" subjects except that for a given $\dot{V}O_2$, % $\dot{V}O_{2\,max}$ and Δf_H, the effects of β blockade and atropine were to raise ΔC (Table 1). The normal (control) relationship of ΔC to $\dot{V}O_2$ and % $\dot{V}O_{2\,max}$ was also displaced to the right, i.e. for given absolute or relative work load the rise in ΔC was lower for the trained compared

Table 1 (continued)

Subject	Gradient %	$\dot{V}O_2$ l · min^{-1}	% $\dot{V}O_{2\,max}$ %	f_H beats · min^{-1}	$\dot{Q}$ l · min^{-1}	C µg/100 ml^{-1}	ΔC	Δf_H beats · min^{-1}	$\Delta\% f_H$ %
β-Blockade									
EZF	0	1.16	28	82	9.7	0.78	0	0	0
	3	–	–	–	–	–	–	–	–
	6	1.62	40	97	12.6	0.90	0.12	15	15
	9	2.02	49	111	14.2	1.02	0.24	29	30
	12	2.45	60	136	14.9	1.62	0.84	54	55
	15	2.94	72	158	17.5	2.76	1.98	76	78
	18	3.43	83	177	19.8	4.68	3.90	95	97
CTMD	0	1.21	35	87	7.5	1.14	0	0	0
	3	1.40	40	92	8.7	1.02	0.12	5	7
	6	1.70	49	103	9.7	1.26	0.12	16	21
	9	1.99	57	119	10.9	1.80	0.66	32	43
	12	2.37	68	138	13.1	2.64	1.50	51	68
	15	2.73	78	153	15.6	5.82	4.68	66	88
JF	0	1.10	36	90	7.5	0.90	0	0	0
	3	1.54	50	92	9.3	0.96	0.06	2	3
	6	1.93	62	108	11.6	1.30	0.40	18	24
	9	2.13	69	127	14.1	1.80	0.90	37	49
	12	2.77	90	160	–	3.78	2.88	70	93
RR	0	1.21	31	87	9.0	2.04	0	0	0
	3	1.64	42	105	10.8	–	–	18	20
	6	1.96	50	118	11.7	2.40	0.36	31	35
	9	2.25	58	138	13.8	–	–	51	60
	12	2.59	66	158	16.6	4.62	2.58	71	81
	15	–	–	167	–	7.50	6.46	80	91
JB	0	2.50	52	113	13.5	1.68	0	0	0
	3	2.73	57	126	15.4	2.34	0.66	13	24
	6	3.21	67	137	17.5	2.94	1.26	24	44
	9	3.76	78	150	19.3	4.50	2.82	37	69
	12	4.18	87	162	–	8.16	6.48	49	91
	14	–	–	167	–	10.08	8.30	54	100

with the untrained subjects. The variance of ΔC in relation to these parameters could only be resolved by considering the exercise cardiac frequency as a percentage change measured from baseline of walking on the level in respect of the maximal attainable f_H in the control and during investigations (Fig. 2). Expressing the Δf_H in this way removes the differences between the $\Delta C/\Delta f_H$ on exercise in the atropine, practolol and control experiments, though the ΔC points obtained in the drug experiments for JB are still slightly higher for given % Δf_H than those obtained on "sedentary" subjects (Table 1 and Fig. 2).

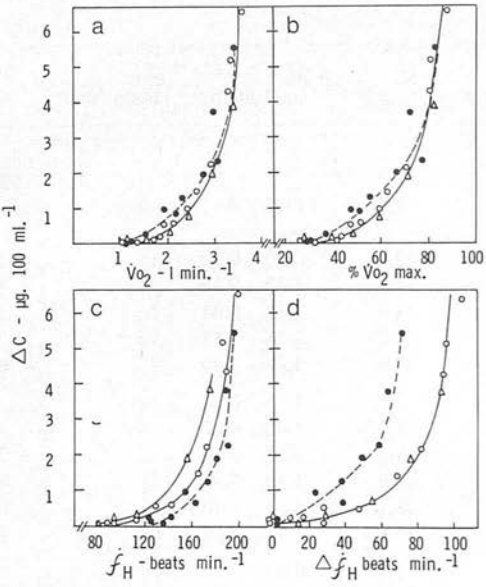

Fig. 1

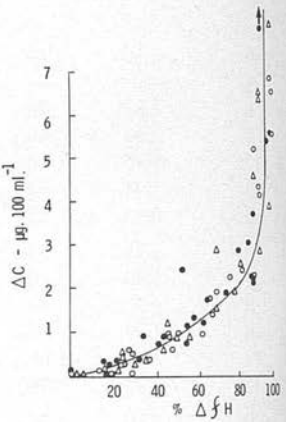

Fig. 2

Fig. 1a–d. The relationship of the change in catecholamine (ΔC) to top left (**a**) oxygen intake ($\dot{V}O_2$), top right (**b**) relative work load expressed as a percentage of $\dot{V}O_{2\ max}$, bottom left (**c**) absolute exercise cardiac frequency (f_H), and bottom right (**d**) change in cardiac frequency calculated from a baseline of work at zero gradient (Δf_H). Subject: EZ. ○ control; △ β blockade; ● atropine

Fig. 2. The change of plasma catecholamine concentration (ΔC) in relation to the percentage change of exercise cardiac frequency (% $\Delta f_{H\ max}$) measured from a baseline of zero gradient and expressed with respect to the maximal attainable change of f_H for a given exercise, i.e. $\frac{f_{H\ ex} - f_{H_0}}{f_{H\ max} - f_{H_0}}$ where $f_{H\ ex}$ = absolute exercise cardiac frequency, f_{H0} = cardiac frequency at zero gradient, and $f_{H\ max}$ = maximal cardiac frequency. ○ control, ● atropine, △ practolol experiments. The arrowed point should be placed at a ΔC of 18.48 μg · 100 ml^{-1}

Discussion

The data from the present experiments confirm the previous findings [4] that for a given type of exercise under normal conditions, ΔC is closely associated with relative aerobic energy expenditure (Table 1, Fig. 1b). In the sedentary subjects the marked rise in ΔC occurs at approximately 60% $\dot{V}O_{2\ max}$, but in the athlete (JB) the ΔC/% $\dot{V}O_{2\ max}$ curve is displaced to the right since the increase in ΔC occurs closer to his maximal aerobic output [10]. The changes in C also reflect the changes in f_H as previously found [4], but the present results show that β blockade reduces the f_H without effecting a change in the $\Delta C/\dot{V}O_2$ relationship (except in the athlete). Thus following practolol administration, the association of ΔC with % $\dot{V}O_{2\ max}$ remains unchanged (Fig. 1b) but for a given f_H, ΔC is increased (Fig. 1d). Atropine had the opposite effects to β blockade; it increased f_H and raised ΔC for a given $\dot{V}O_2$ and % $\dot{V}O_{2\ max}$. The rise in ΔC was less than the change in f_H; so that the $\Delta C/f_H$ curve

was displaced to the right of the normal line linking the two variables (Fig. 1d). These data confirm our view [4] that the normal relationship between changes in plasma catecholamine levels and cardiac frequency is not causal but merely an association between two independent variables. The differences in the $\Delta C/f_H$ behaviour with drug infusion could not be reconciled by considering $\dot{Q}$, since the average values of cardiac output for a given $\dot{V}O_2$ (Table 1) were similar in the control and atropine experiments while β blockade effected only a small decrease in $\dot{Q}$. Similar results for the effects of atropinisation and β blockade on the circulatory system have been obtained by Ekblom et al. [8]. The inter- and intra-subject differences of ΔC (Table 1) in relation to the circulatory system could only be reduced by considering, not f_H per se, but Δf_H measured from a baseline of walking at 0% gradient and expressed as a percentage of the maximal Δf_H attainable by the individual in the different types of work. This latter procedure essentially removed the small differences in the Δf_H due to age; it should be noted that our subjects' mean age was 35.6 yrs with a range of 26–46 yrs, and the maximal cardiac frequency attained in control and atropine experiments varied from 182–200 beats $\cdot$ min^{-1}. When ΔC is plotted against % Δf_H the relationship is shown to be curvilinear ($r = 0.93$; $P < 0.001$) and accounts for 82% of the total variance of the two variables for all experiments in the sedentary subjects. In the athlete the same appears to be true, but his points, particularly for atropine and β blockade, are displaced to the left of the "sedentary" $\Delta C/\%\ \Delta f_H$ line.

Both in this and a previous study [4] the catecholamines measured in our blood samples have been composed almost entirely of nor-adrenaline. The only known cardiovascular functions of noradrenaline are to increase constrictor tone and stimulate the β receptors of the myocardium. We have therefore argued that the association of ΔC with a circulatory variable must reflect either one or both of these factors. From the results of the present experiments we can now go further. Following the administration of practolol, the β cells of the heart were (at least partially) blocked, yet the ΔC for a given $\dot{V}O_2$ remained unchanged in all subjects except JB, and the $\Delta C/\%\ \Delta f_H$ relationship was unaffected. It would thus seem reasonable to suggest that the changes in ΔC during exercise (particularly at intensities > 60% $\dot{V}O_{2\ max}$) affect the f_H indirectly due to changes in vasomotor tone elicited by release of nor adrenaline from sympathetic nerve endings within the blood vessels.

It is well known that exercise induces constriction [2] principally at two sites, the skin and splanchnic region. The vasoconstriction which occurs in "the skin" during short term exercise seems to be proportional to the absolute work rate [1] and is not affected by α adrenergic blockade with phentolamine [18]. This indicates that peripheral vasoconstriction is due to localised symphathetic release of catecholamine by vasomotor nerve endings. Undoubtedly the catecholamine levels in our blood samples in part reflect this aspect of blood flow regulation. However, in these experiments it will be noted that ΔC is a curvilinear function of both relative and absolute work loads so that a theory based solely on an increased vasoconstrictor tone in the skin blood vessels is not consistent with our data. On the other hand, Rowell and his co-workers [15, 16] have shown that whenever blood is displaced from the splanchnic region, cardiac frequency rises, while the increase in vasoconstrictor tone is proportional to relative work load. We therefore feel that the changes we have observed in ΔC are primarily (but not exclusively) related to the closure of the

splanchnic circulation associated with relative work load. This closure is probably effected by nor adrenaline release from sympathetic nerve endings located in visceral blood vessels; it seems unlikely that the adrenal gland makes any significant contribution to the release of ΔC during exercise, since adrenolectomy in animals [7] and renal catheterisation in man [19] show that the adrenal glands contribute little to the pattern of adaptation to exercise.

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Temperature regulation during severe exercise with some observations on effects of skin wetting

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DAVIES, C. T. M., J. R. BROTHERHOOD, AND E. ZEIDIFARD. *Temperature regulation during severe exercise with some observations on effects of skin wetting*. J. Appl. Physiol. 41(5): 772-776. 1976.—The thermoregulatory responses to severe exercise of 1-h duration have been studied in 11 healthy subjects, and the effects of skin wetting have been examined in 6 of them. Measurements in the laboratory included metabolic (M) and total (H) heat production, rectal (T_{re}) and mean skin (T_{sk}) temperatures, evaporative sweat loss (E), and peripheral tissue conductance (K). In addition T_{re} and changes in body weight were measured in 18 competitors following a 20-km road race run at approximately 90% of their maximal oxygen intake ($\dot{V}O_{2\,max}$). The results showed that T_{re} was curvilinearly related to $\%\dot{V}O_{2\,max}$. In contrast T_{sk} was independent of M, H, E, and $\%\dot{V}O_{2\,max}$, and reached similar and constant values at the end of exercise in all subjects. E was appropriate for a given H, but K was lower in the sedentary subjects than in the athletes. Skin wetting had no effect on T_{re} but reduced E by 10%. It is suggested that the pattern of T_{re} rise in exercise reflected the nature of the controlling system and was not due to a failure of the heat-dissipating mechanisms.

rectal temperature; tympanic temperature; sweating; conductance; maximal aerobic power.

IT IS WELL KNOWN that, in a given individual, body temperature (T_c) during exercise rises to a level which is proportional to the oxygen intake ($\dot{V}O_2$) independently of a wide range of ambient temperatures (13, 15). However, when groups of individuals are studied, it has been shown (1, 22) that the intersubject variability of T_c is reduced if it is related not to absolute but to relative metabolic rate (expressed as % of maximal oxygen intake – $\dot{V}O_{2\,max}$). The relative work load and state of acclimatization govern the (prescriptive) zone of "environmental independence" of T_c (10). From the work of Saltin and Hermansen (22) the T_c-$\%\dot{V}O_{2\,max}$ relationship would appear to be linear but their study was limited to relative work levels at or below 75% $\dot{V}O_{2\,max}$. Clearly their data cannot be used outside this range, since, if extrapolated, they suggest that at a 100% $\dot{V}O_{2\,max}$ body temperature would only rise to 39.6°C. This is not consistent with the data recorded on marathon athletes, in whom temperatures of ~41°C have been observed at the end of a competition (17).

To extend the work of Saltin and Hermansen (22) we have studied 11 subjects under standardized laboratory conditions at levels of exercise ranging from 45 to 86% $\dot{V}O_{2\,max}$ and a group of athletes at the finish of a 20-km road race demanding approximately 90% $\dot{V}O_{2\,max}$. We also present some observations on the effects of skin wetting (sponging) on body temperature during severe exercise.

SUBJECTS AND METHODS

Eleven subjects were studied in the laboratory and 23 athletes were investigated in the field. The physical characteristics of the laboratory group are given in Table 1. Two of the subjects were athletes; *subject 5* completed in 400/800 m events and *subject 8* was an endurance (road) runner. *Subjects 3, 4, 7, 10,* and *11* were active but at the time of measurement were not in training, although *subject 4* was a physical education student and the others did run occasionally on weekends. The remaining subjects were of sedentary habits.

In the laboratory the subjects were studied under standardized conditions on a motor-driven treadmill. They were weighed nude, and in shorts and tennis shoes before exercise, using a balance accurate to ±5 g, and then they walked (or ran) at constant speed for 1 h at different gradients, to produce a range of exercise intensities covering the level of each subject's work capacity. The environmental conditions were $T_{db} = 21.0 \pm 1.4$°C, $T_{wb} = 16.8 \pm 2.2$°C, and the relative humidity = 48%.

A total of 54 experiments were performed. Rectal (T_{re}) and tympanic (T_{ty}) temperatures were measured at rest and continuously throughout exercise, by means of a thermistor probe inserted inside the rectum 8 cm above the anal sphincter, and a zero temperature gradient thermometer (9) inserted into the auditory canal close to the tympanum.

Subsequent analysis showed that although T_{re} was on average 0.14–0.25°C higher than T_{ty} (cf. Nielsen and Nielsen (14)) the two variables were so closely associated

$$T_{ty}\ (°C) = 0.905\ T_{re}\ (°C) + 3.49 \qquad (r = +0.93;\ P < 0.001)$$

that either could be used as an index of core temperature without material loss of accuracy. For convenience therefore in the present analysis we have used T_{re}.

Skin temperature was measured at rest and at approximately 15-min intervals during exercise using a thermocouple bead probe mounted in a plastic applicator, placed at 13 sites, on the hand, upper and lower

TABLE 1. *Physical characteristics*

Subj No.	Type	Age, yr	Wt, kg	Ht, cm	$\dot{V}O_{2\,max}$ l·min^{-1}	$\dot{V}O_{2\,max}$ ml·kg^{-1}·min^{-1}
1	Sedentary	22	64.3	175.2	3.20	49.7
2	Sedentary	21	71.5	178.7	3.30	46.2
3	Active	41	63.9	178.0	3.50	54.8
4	Active	26	62.0	172.5	3.41	55.0
5	Athlete	21	78.9	180.0	5.40	68.4
6	Sedentary	31	72.8	178.9	3.20	44.0
7	Active	30	62.6	168.8	3.90	62.3
8	Athlete	35	63.9	174.0	4.80	75.1
9	Sedentary	24	64.0	165.9	2.20	34.3
10	Active	26	71.8	173.0	4.10	57.1
11	Active	21	70.0	189.4	4.16	59.4
Mean		27.1	67.8	175.8	3.74	55.12
±SD		±6.5	±5.5	±6.3	±0.87	±11.47

arm, forehead, pectoral, sternum, abdomen, scapula, lumbar, anterior and posterior thigh, and anterior and posterior calf. The surface recordings were weighted after the method of Hardy and DuBois (8), and the average value was taken as the mean body skin temperature ($\bar{T}_{sk}$). $\dot{V}O_2$ was measured using an open-circuit technique; CO_2 and O_2 content of expired air was measured with analyzers regularly calibrated with gas concentrations known from Lloyd-Haldane analysis. Cardiac frequency (f_H) was measured from chest electrodes coupled to a differential amplifier and linear instantaneous rate meter. Following exercise the subjects were immediately weighed clothed, and then dried and reweighed nude. From the recorded weight losses the total and evaportive sweat losses were determined. From the raw data the metabolic (M) and total heat production (H), heat storage (S), evaporative sweat loss (E, corrected for respiratory and metabolic water losses), and body conductance (K) were calculated in the standard way. In six subjects the experiments at approximately 75% $\dot{V}O_{2\,max}$ were repeated but in addition the skin was sponged with water at the appropriate skin temperature at regular intervals throughout the exercise.

In the field, observations were made of the weight losses and running speeds of a small sample of 23 of a total of 152 athletes who took part in a 20-km road race. In 18 of the 23 athletes, rectal temperatures were recorded immediately after they had crossed the finishing line. Before and after the race the athletes were weighed on portable beam balances accurate to ± 50 g. Rectal temperature was taken with a mercury in glass thermometer inserted 8 cm inside the rectum for at least 2 min. Competitors' time and speed were noted, and from these and the weight change observations, an estimate of aerobic energy expenditure, total heat production, and evaporative sweat loss were made using the methods of Pugh (16, 17). The athletes' $\dot{V}O_{2\,max}$ was estimated from his best 5,000-m time. The day of the race was calm (wind speed = 1.46 m·s^{-1}) and overcast with occasional sunny intervals. The T_{db}, T_{wb}, and relative humidity were 15.1°C, 10.4°C and 58%, respectively.

RESULTS

At all levels of exercise in the laboratory and the field a large intersubject variability of T_{re} in relation to $\dot{V}O_2$ was found (Fig. 1). The variance associated with T_{re} and $\dot{V}O_2$ could not be significantly reduced by considering the body weight or surface area of the subjects; this could only be achieved by taking into account the individual $\dot{V}O_{2\,max}$ and expressing $\dot{V}O_2$ in relative rather than absolute terms. However, the relationship between T_{re} and $\%\dot{V}O_{2\,max}$ is not linear (Fig. 2); and it is more adequately described by a quadratic equation of the form

$$T_{re}\ (°C) = 37.25 - 0.00264\ (\%\dot{V}O_{2\,max}) + 0.00037\ (\%\dot{V}O_{2\,max})^2$$

In the laboratory experiments $\bar{T}_{sk}$ was independent of M (Fig. 1), and E, both in the field and laboratory, was proportional to H (Fig. 3). The calculated K values for given $\dot{V}O_2$ of 2 l·min^{-1} were higher in the athletes compared with the more sedentary subjects.

The effect of skin wetting (Table 2) was to reduce E by approximately 10% ($P < 0.05$) and produce a small fall (0.02°C) in T_{re} but this did not materially change the relationship given in Fig. 2.

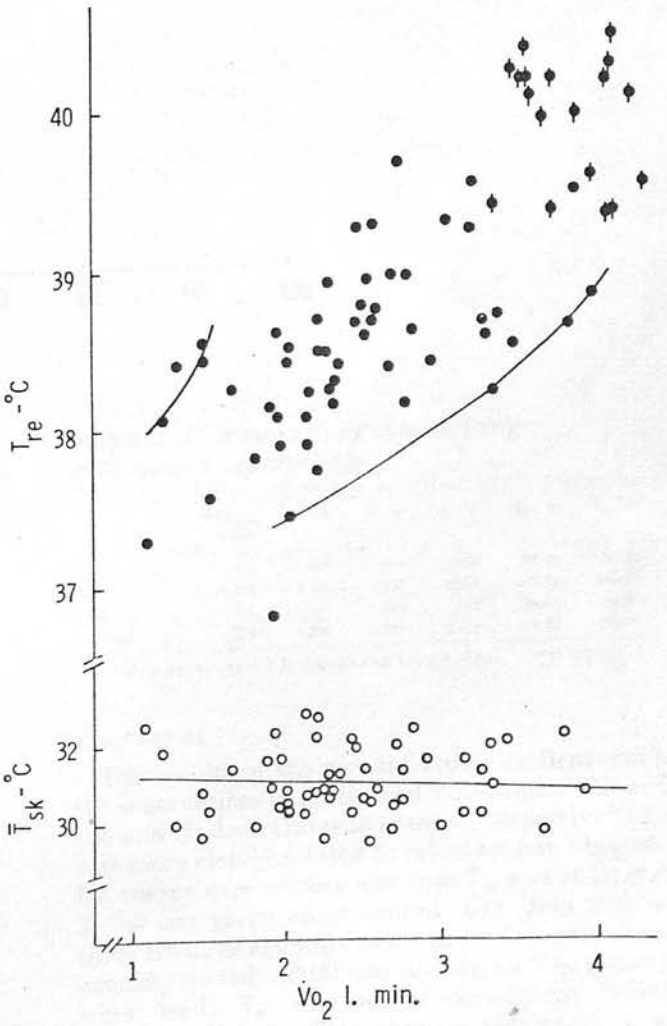

FIG. 1. Rectal temperature (T_{re}) and mean skin temperature ($\bar{T}_{sk}$) in relation to oxygen intake ($\dot{V}O_2$). Lines join points for subject with lowest (*no. 9*) and highest (*no. 8*) $\dot{V}O_{2\,max}$ (see Table 1). Laboratory (●) and field (◉) values.

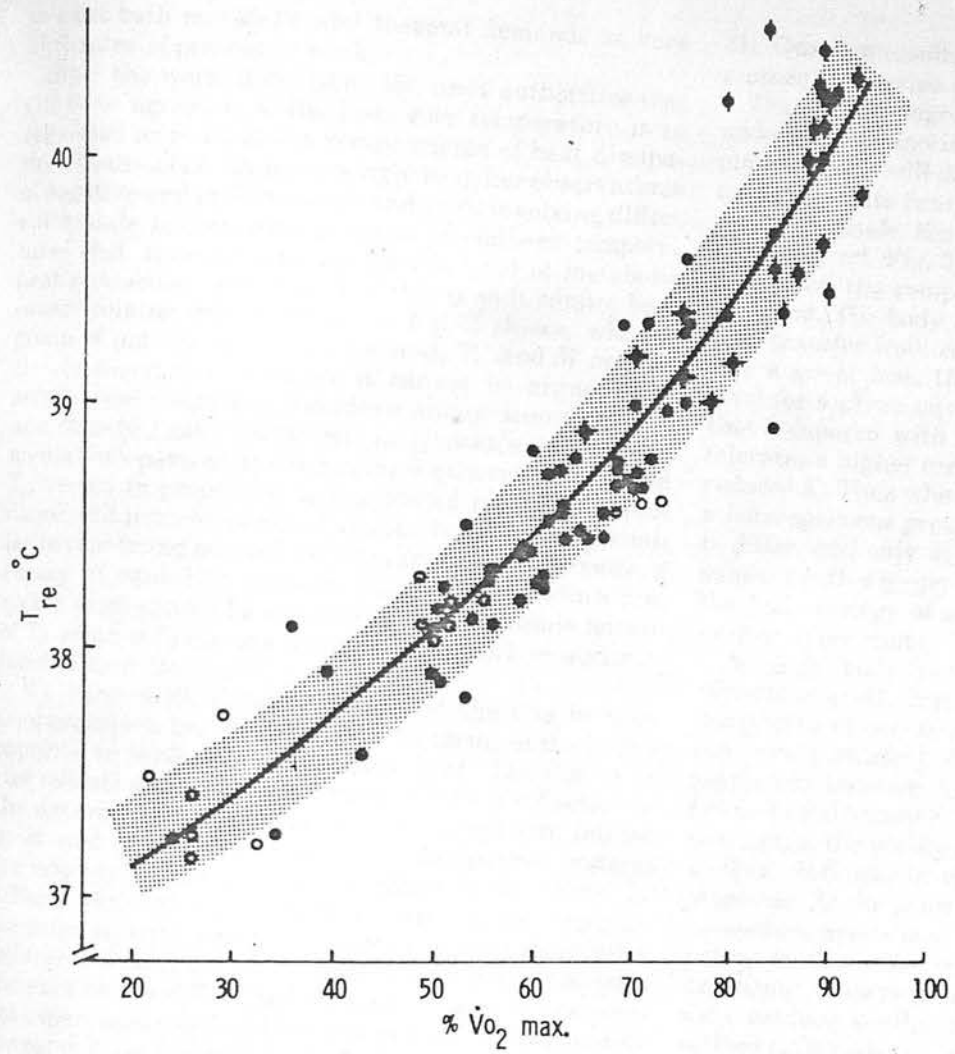

FIG. 2. Relationship of rectal temperature to $\dot{V}o_{2\,max}$. Present laboratory (●), field (◐) data, and skin wetting (△). Data of Saltin and Hermansen (22) (○). Curved (- - -) line is of the form of T_{re} (°C) = 37.25 − 0.00264% $\dot{V}o_{2\,max}$ + 0.00037 (%$\dot{V}o_{2\,max}$)². Solid lines represent ± 1 SD of observed points. Analysis of covariance reveals that quadratic equation of this form is of significantly ($P < 0.01$) better fit than linear model.

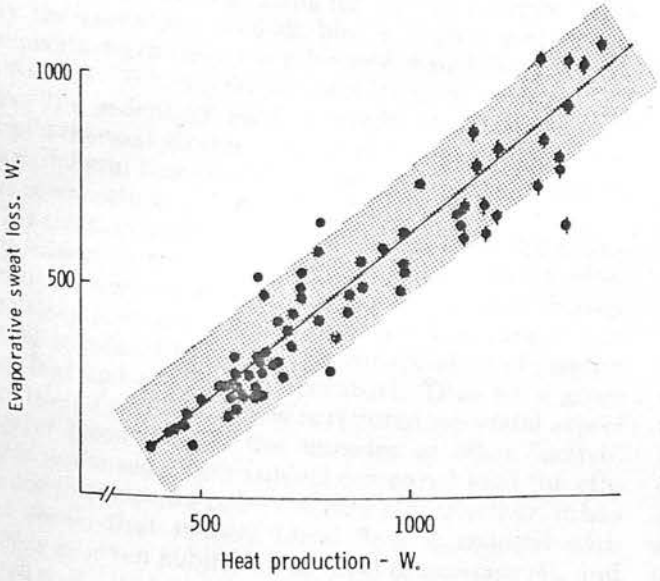

FIG. 3. Relationship of evaporative sweat loss (E), corrected for metabolic and respiratory water loss, to total heat production (H). Regression equation is of the form: E (W) = − 184.82 + 0.793 H (W); γ = + 0.88; SD = ± 82.0.

TABLE 2. *Comparison of skin wetting with control experiments*

	%$\dot{V}o_{2\,max}$, units	M, W	H, W	E, W	T_{re}, °C	$\bar{T}_{sk}$, °C	K, W·°C·m⁻²
Skin wetting	74.0	970	821	490	39.10	30.39	43.0
(n = 6)	±6.5	±206	±121	±214	±0.36	±0.61	±12.6
Controls	73.6	967	820	545*	39.11	30.85	42.1
(n = 6)	±6.7	±220	±199	±234	±0.17	±0.88	±16.5

Values are means ± S.D. Significance (paired t-test): * $P < 0.05$.

DISCUSSION

The results of the present study confirm and extend the observations of Saltin and Hermansen (22) and Nielsen and Nielsen (14) who showed, respectively, that T_{re} was more closely related to relative than absolute aerobic energy expenditure and that $\bar{T}_{sk}$ was independent of M for any given environment. Our data suggest that these findings are valid over the whole range of exercise capacity an individual can sustain for 1 h, but at higher work loads T_{re} becomes a curvilinear function of %$\dot{V}o_{2\,max}$ (Fig. 2). This phenomenon could be seen to represent a failure of the heat-dissipating mechanisms or alternatively as part of a common strategy designed

to meet both metabolic and thermal demands at very high rates of prolonged work.

Since the work of Nielsen (15), most authorities (see (7)) have agreed that the body core temperature is so regulated as to meet the requirements of heat dissipation. Nielsen (12) argues strongly from her observations of negative and positive work and work involving different muscle masses over a range of ambient temperatures that T_c is actually set by the level of metabolic heat production. But clearly if this is so it cannot be a causal relationship, because as Fig. 1 shows, when a group of individuals are considered, T_{re} and M are not closely associated. Further it cannot be argued that aerobic heat liberation provides a unique stimulus to T_{re} and thus to heat dissipation. In Nielsen's own experiments with passive heating using diathermy (14), E and T_{re} varied in proportion to summated rate of heat produced and induced passively in the body in a way similar to that found previously for exercise. Furthermore, a theory of causality between central temperature and metabolism cannot be consistent with the labile nature of T_c when subjects are habituated to work or acclimatized to heat (5).

We agree with Benzinger (2) that the rise in body temperature is better understood in terms of the body's capacity to produce and dissipate heat. The degree of rise (offset) of T_c during exercise will be a reflection of the nature of the (proportional) control system, related to M and the power of the heat-dissipating systems (principally E and the flow of blood to the skin). To achieve thermal equilibrium within a given environment the sedentary subject and athlete must maintain a balance between heat delivered to and dissipated from the skin by blood flow and E, respectively. In the present experiments each subject maintained approximately the same $\bar{T}_{sk}$ at the end of 1 h of exercise (Fig. 1) over the range of this work capacity, and independently of H, M, or his state of training. Thus for a given exercise intensity the sedentary subject has a higher core-to-skin temperature gradient and a lower K which is indicative, as Nielsen (12) has inferred, of a lower peripheral blood flow. The sedentary subject would therefore seem to adopt a thermal strategy which minimizes blood flow to the peripheral tissues at the expense of a higher central core temperature.

The strategy appears to be an attempt by the sedentary subject to meet the competing demands for blood flow to the working muscles, and to the skin for thermoregulatory purposes. It is well known that cardiac output (Q) is related to M (3), and independent of relative work load and ambient temperature. Thus for a given work load, if skin blood flow is reduced we would expect a larger blood flow to the muscles or other "active" organs in the sedentary subject compared with the athlete and this appears to be the case (18). Further, it has been shown that muscle blood flow is reduced with training at given submaximal level of exercise (4), and Rowell et al. (19) have shown that the degree of vasoconstriction in the splanchnic region is related to relative work load so that for a given Q or M, sedentary subjects are relatively more vasoconstricted than the athlete (20, 21). Our own results on the changes in plasma catecholamines in exercise would support this conclusion (6).

The rise (or degree of offset) of T_{re} during exercise is undoubtedly associated with M (Fig. 1), but its final plateau value will depend on the capacity of the individual to dissipate heat both by evaporative and nonevaporative channels. Essentially, provided E is adequate for a given H (cf. Fig. 3) the thermal strategy adopted will depend on the competing interests of muscle and skin for blood. The body attempts to balance the convective heat transfer from core to periphery against the evaporative sweat loss, thus maintaining $\bar{T}_{sk}$ at a constant level for a given environmental condition. This means that compared with the athlete the sedentary subject tolerates a higher core temperature for a given M and a reduced K. Thus when T_{re} is compared at the same M in a heterogeneous group of subjects it would be expected to differ, and only at very high absolute work loads in subjects with a larger cardiovascular capacity would the thermal strategy of a sedentary subject become necessary or appropriate.

At high body temperatures during severe work, Wyndham et al. (23) have argued that the two heat-dissipating channels (E and K) become saturated, i.e., the curves relating E and K to T_{re} tend toward an asymptote between 38.9 and 39.2°C. At temperatures below 38.5°C where E and K operate at the most sensitive range, the ability of an individual to dissipate heat is then well within the capacity of the two thermal processes. At the point of saturation, Wyndham and his co-workers argue that since the capacity of heat-dissipating mechanisms becomes exhausted Δ H must rise, producing an increase in T_{re}. Certainly plots of our own data produce similar curves of K and E to those described by Wyndham, so that it could be argued that the rise in T_{re} is due to a saturation in the capacity of the heat-regulatory mechanisms, causing an increase in body heat stores. However, we do not feel that T_{re} is the appropriate variable to relate to either E or K. Our view is that E should be related to H, and K to either M or H. If this is done (Fig. 3) then it can be seen from our results that E is closely related to H at all levels of work for both the field and laboratory experiments. Though in this context the reader should compare the results of Pugh, Corbett, and Johnson (17), who found that the sweating mechanism was only 43% efficient in their study of the Witney marathon race. Further, our skin wetting experiments (Table 2) were without effect on T_{re}; but in accord with the findings of Nadel and Stolwijk (11) they did reduce E. From these data it would be difficult to argue a failure or saturation in the evaporative heat-dissipating mechanisms. Again it is our view that the rise in T_{re} at higher levels of work offsets a decline in blood flow to the skin. At these levels of work exercise can be maintained provided the evaporation of sweat is adequate to maintain a constant $\bar{T}_{sk}$. The price to be paid for a partially vasoconstricted skin is the toleration of a raised T_{re}. At high relative work loads a low $\bar{T}_{sk}$ is an important factor for maintaining exercise, and it is here that the individual may become very sensitive to environmental conditions. Small changes in

radiant heat, for example, could have a deleterious effect on performance. This phenomenon has been alluded to by Pugh et al. (17) who found that performance in marathon runners was related to their ability to sustain and tolerate a high T_{re} and could be critically dependent on environmental conditions in the presence of direct solar radiation.

A constant $\bar{T}_{sk}$ in exercise under set environmental conditions must imply (as first pointed out by Nielsen (12)) that heat transfer to and from the skin by convection and evaporation, respectively, must not only be balanced but finely coordinated. In a thermally neutral environment if $\bar{T}_{sk}$ is held constant then E must be a function of H to meet the requirements of the heat balance equation (Fig. 3).

Similarly if the Fick principle is applied to heat transport, the blood flow to the periphery must be a function of the temperature gradient between the arterial (core) and (peripheral) venous blood. Therefore if for a given $\%\dot{V}O_{2\,max}$ T_{re} is held at a constant level (Fig. 2) the amount of blood in the periphery will be associated with $\bar{T}_{sk}$. The $\bar{T}_{sk}$ in turn will depend on E, T_a, and the convective transfer of heat to the periphery. It is the interaction between these variables and the maximal aerobic power output of the individual which will ultimately set the upper limits of thermal regulation and physiological performance in prolonged hard work.

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Functional and structural changes after disuse of human muscle

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Summary

1. Seven patients who had suffered unilateral eg fracture were studied after removal of mmobilizing plaster casts.

2. Leg volume measured anthropometrically vas reduced by 12% in the injured leg (5·68 ± ·05 litres) compared with the uninjured (6·43 ± ·87 litres). Associated with this loss was a imilar reduction in the net maximum oxygen ptake achieved in one-leg cycling, from 1·89 ± ·21 l/min in the uninjured leg to 1·57 ± 0·18 min in the injured.

3. Measured by a percutaneous needle biopsy echnique, a reduction of 42% was found in the oss-sectional area of the muscle fibres ampled from the vastus lateralis of the injured ompared with the uninjured leg.

4. Staining for myosin adenosine triphospha-se activity showed that both type I and II ores were affected, being reduced respectively om 3410 to 1840 μm^2 and from 3810 to 2390 n^2 cross-sectional area.

5. Possible reasons and implications are dis-ssed for the discrepancy between the magni-de of the difference observed in the gross easurement of leg function (maximum oxygen ptake) and structure (leg volume) as compared ith the cellular level (cross-sectional fibre ea).

ey words: atrophy, muscle, oxygen uptake.

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Introduction

Atrophy of the affected limb and loss of muscle power follows bone fracture and subsequent immobilization. Years of experience have enabled the rehabilitation professions to develop empirical programmes to reverse these changes. However, the efficacy of such programmes may be further improved if we can increase our understanding of the atrophic response to disuse in human muscle.

Recent studies showed that 15 weeks immobilization in a long-leg plaster cast after fracture reduced the fat-free volume of the affected leg by 12%, which was accompanied by a similar fall in the maximum oxygen uptake ($\dot{V}O_{2max.}$) achieved with one-leg pedalling (Davies & Sargeant, 1975a,b). However, it was not known how far these changes in gross structure and function were reflected at a cellular level within the affected muscles.

Since the work of pedalling is performed mainly by the leg extensors (A. J. Sargeant & C. T. M. Davies, unpublished work) needle biopsy was used (Edwards, Maunder, Lewis & Pearse, 1973) to study fibre atrophy in the quadriceps femoris muscle and to compare this with measurements of the gross leg volume and maximal oxygen uptake of patients recovering from unilateral leg fracture.

Methods

The patients, seven otherwise healthy young servicemen, had tibia and fibula fractures of one leg, treated by immobilization of the injured

limb in a long leg plaster, for a mean period of 131 days (range 53–213 days). Our measurements were made from 0 to 42 days after effective mobilization (calculated from the time the patients were assessed as being weight-bearing on the affected leg after removal of the plaster). We also studied eight healthy male subjects who had no recent leg injuries. All subjects, both normal and patients, were volunteers and gave their free informed consent.

The investigation and the techniques involved were approved by the Research and Ethics Committee of the Royal Post-graduate Medical School, Hammersmith Hospital.

The volume of each leg (muscle plus bone) was determined anthropometrically (Davies & Sargeant, 1975c). The estimation of the maximum oxygen uptake ($\dot{V}O_{2max.}$) in one-leg cycling has been described in detail (Davies & Sargeant, 1975a). The subjects pedalled a fixed-wheel stationary bicycle ergometer in a continuous progressive test spanning the subjects' working capacity from zero up to maximum in four or five work loads each lasting 5 min. $\dot{V}O_{2max.}$ was measured by the Douglas bag technique over the last minute of the final 3 min of work as the subject was encouraged to pedal at maximal effort. If this could be sustained the load was increased and $\dot{V}O_2$ measurements were repeated. Owing to the difficulty of obtaining maximal measurements in one-leg exercise which meet the 'plateau' criterion developed for two-leg exercise, it was sometimes necessary to take duplicate measurements on subsequent days. $\dot{V}O_{2max.}$ was expressed after subtracting the $\dot{V}O_2$ during pedalling at a constant speed against zero load (Hill, 1965; Whipp & Wasserman, 1969).

Needle biopsies were taken from the lateral part of the quadriceps muscle at the junction of the distal and middle thirds of the thigh, with a Bergström needle inserted through a 4 mm skin incision (Bergström 1962, 1975; Edwards, 1971). The biopsy was prepared as previously described (Edwards *et al.*, 1973). Transverse 10 μm frozen sections were stained for myosin adenosine triphosphatase activity at pH 9·4 and after preincubation at pH 4·3 (Hayashi & Friemau, 1966; Brooke & Kaiser, 1969) to identify type I and type II muscle fibres. The relative frequency of the two fibre types was counted in each biopsy and the mean cross-sectional area of each type of fibre calculated from measurement of the 'lesser fibre diameter' in 50–100 fibres (Dubowitz & Brooke, 1974), to give the overall mean cross-sectional fibre area for each biopsy.

Results

Mean results ± SD are given.

Leg volume and oxygen uptake

After immobilization the total leg volume (which is muscle plus bone) measured anthropometrically was on average 12% less in the injured (5·68 ± 1·05 l) compared with the uninjured (6·43 ± 0·87 l) leg. The loss was similar in both upper and lower leg measurements (Table 1).

The $\dot{V}O_{2max.}$ achieved in one-leg cycling was also reduced from 1·89 ± 0·21 l/min in the uninjured to 1·57 ± 0·18 l/min in the injured leg. All values of $\dot{V}O_{2max.}$, except in subject no. 3, fell within the limits for the relationship leg vol./$\dot{V}O_{2max.}$ previously reported (Davies & Sargeant, 1975a), for both injured and uninjured legs (Fig. 1).

Changes in muscle fibres

The frequencies of type I and type II fibres (mean 61 and 39 ± 10% respectively) are not significantly different between the injured and uninjured legs. Both type I and type II fibres of the injured leg show a significant ($P<0·01$) and similar reduction in the mean cross-sectional area, when compared with the uninjured leg, type I fibres being reduced from 3410 ± 530 μm^2 in the uninjured to 1840 ± 410 μm^2 in the injured leg, and type II fibres from 3810 ± 940 μm^2 to 2390 ± 910 μm^2. A combined mean fibre area calculated for each leg, taking into account the frequency and mean area of both fibre types, shows on average a reduction of 42%, from 3570 ± 630 μm^2 in the uninjured to 2080 ± 550 μm^2 in the injured leg (Fig. 2). The degree of atrophy, as indicated by the mean fibre area, was correlated ($r = 0·82$, $P<0·05$) with the length of time (from 0 to 42 days) that the patients had been weight-bearing (Fig. 3).

The percentage reduction in mean fibre area of the injured leg is proportionally greater than is indicated by anthropometric assessment

TABLE 1. *Structure and function in injured and uninjured legs in seven patients and eight normal subjects*

ui = uninjured leg; i = injured leg; s = stronger leg; w = weaker leg.

Patients											Normal subjects					
No.	Age (years)	Time immobile (days)	Time wt. bearing (days)	Leg volume (l)		$\dot{V}O_{2max.}$ net (l/min)	Fibre type frequency (%)		10^{-2} × Mean fibre area (μm^2)		No.	Age (years)	Fibre type frequency (%)		10^{-2} × Mean fibre area (μm^2)	
				Total	Upper		I	II	I	II			I	II	I	II
1	19	54	28 ui	6·96	4·08	1·86	67	33	39	42	N1	25 s	72	28	49	60
			i	6·37	3·73	1·68	62	38	22	27		w	64	36	46	63
2	18	53	7 ui	5·33	3·11	—	55	45	27	30	N2	20 s	58	42	37	43
			i	5·00	2·90	—	62	38	13	23		w	62	38	36	37
3	29	213	0 ui	5·46	3·18	1·88	66	34	30	27	N3	22 s	45	55	22	27
			i	4·45	2·47	1·44	79	21	14	7		w	57	43	19	30
4	26	183	14 ui	6·20	3·60	1·66	54	46	37	36	N4	22 s	53	47	33	37
			i	5·19	3·01	1·42	38	62	16	17		w	58	42	27	27
5	30	180	42 ui	7·31	3·99	2·28	59	41	29	37	N5	32 s	64	36	33	35
			i	6·88	3·74	1·87	58	42	22	29		w	75	25	30	36
6	18	85	30 ui	7·64	4·78	1·79	71	29	37	39	N6	33 s	78	22	27	36
			i	7·03	4·16	1·57	51	49	23	32		w	50	50	24	27
7	20	150	14 ui	5·92	3·49	1·87	64	36	40	56	N7	23 s	70	30	42	58
			i	4·87	2·79	1·41	63	37	19	32		w	70	30	45	52
											N8	27 s	56	44	32	40
												w	57	43	37	37

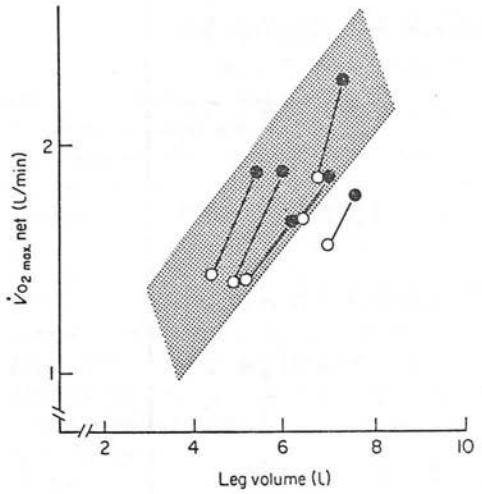

FIG. 1. Relationship of $\dot{V}O_{2max.}$ (net) of one-leg exercise to leg volume (muscle plus bone). Data points are given for patients' injured (○) and uninjured (●) legs, linked with a bar. The stippled area gives the confidence limits of the relationship in normal subjects (Davies & Sargeant, 1974).

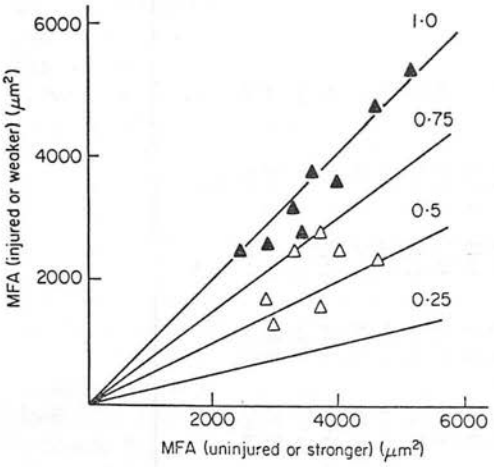

FIG. 2. Mean fibre area (MFA) of the injured (or weaker) leg in relation to that of the uninjured (or stronger) leg for the patients (△) and the normal subjects (▲).

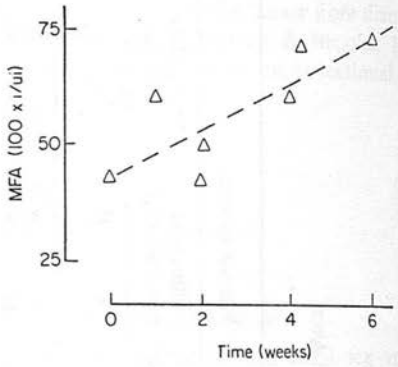

FIG. 3. Degree of muscle fibre atrophy in relation to the time spent weight-bearing after removal of plaster cast. Mean fibre areas (MFA) of the injured (i) legs are expressed as percentages of those in the uninjured (ui) legs. The regression line (– – –) is $y = 43{\cdot}4 + 5{\cdot}12x$; $r = 0{\cdot}82$.

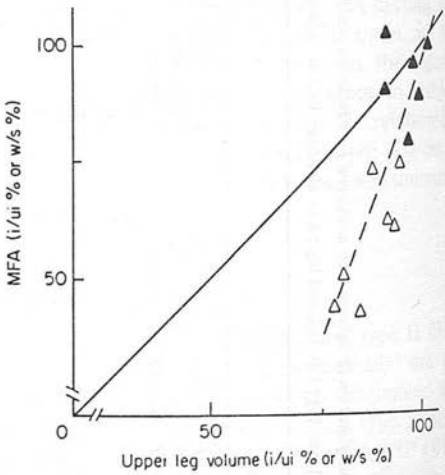

FIG. 4. Degree of muscle fibre atrophy (MFA, see Fig. 3) related to the reduction in anthropometric estimation of the thigh (muscle plus bone) volume. △, Patients [injured leg measurements (i) are expressed as a percentage of the uninjured (ui)]; ▲, normal subjects [weaker leg measurement (w) is expressed as a percentage of the stronger (s)]. The regression line for the combined data (– – –) is $y = 2{\cdot}287x - 134{\cdot}2$; $r = 0{\cdot}78$.

of leg volume (Fig. 4). Similarly, since $\dot{V}O_{2max.}$ is related to leg volume (Fig. 1) the reduction in mean fibre area is also proportionally greater than the reduction of $\dot{V}O_{2max.}$ when the injured leg is compared with the uninjured (Table 1).

Discussion

The atrophic response of human muscle to disuse is a common clinical observation, but there are surprisingly few data available on the functional and structural implications of disuse atrophy in man. In recent investigations (Davies & Sargeant, 1975a, b, c) aspects of disuse atrophy were examined in patients who had had one leg immobilized in a plaster cast after unilateral leg fracture. This enabled direct comparisons between the injured and the uninjured legs after a known period of im-

mobilization and muscle disuse. By the same method we have made direct comparisons of the changes in the muscle fibres. Unlike earlier studies on disuse atrophy, where the biopsy samples were taken at the time of surgical intervention in an affected limb (Patel, Razzak & Dastur, 1969; Edstrom, 1970), by use of percutaneous needle biopsy we obtained samples from both affected and unaffected limbs.

The changes in both $\dot{V}O_{2max.}$ of one-leg exercise and leg volume (muscle plus bone) in our patients (Fig. 1) confirm the previous findings, a fall of approximately 15% in both variables being indicated when the injured is compared with the uninjured leg.

The frequency and size of muscle fibre types in the patients' uninjured leg were similar to those for the healthy male control subjects. There was no systematic difference in the frequency of fibre types between the two legs in the patients, and the variation was no greater than found in bilateral samples from the normal control subjects. The cross-sectional areas of both fibre types were significantly reduced in the injured legs of all the patients when compared with their own uninjured legs (Table 1, Fig. 2). In six of the seven cases, type I fibres showed relatively greater atrophy than type II fibres; thus the mean reduction in type I fibres was 46% compared with 37% in type II fibres. Edstrom (1970) observed a similar tendency for red muscle fibres to atrophy more than white. However, he studied patients with long-term (~2 years) knee-joint dysfunction who cannot be considered comparable with our patients.

These findings differ from the predominantly type II fibre atrophy in the muscle of patients with a variety of non-muscular disorders, e.g. osteomalacia, chronic alcoholism, corticosteroid overdose etc. (Dubowitz & Brooke, 1974). The suggestion, therefore, that type II fibre atrophy in these conditions may merely reflect their reduced habitual activity with relative disuse, due to pain or to general ill-health, may be open to question.

We obtained biopsies from 0 to 42 days after the patients started weight-bearing, and were able effectively to exercise the atrophied limb. Not surprisingly we found a significant correlation (Fig. 3) between the overall degree of fibre atrophy and the time that the patients had been weight-bearing. However, our study includes patients with periods of preceding immobilization ranging from 53 to 213 days, and more, particularly longitudinal, information is needed on the time-course of the recovery process.

The measurements of cross-sectional area of individual fibres indicate that atrophy in the quadriceps muscle is much greater than would be suspected from measurements of either the total leg or thigh volume (Fig. 4), and might imply that when thigh volume was reduced by approximately 50% fibre area would be zero. Part of this discrepancy may be accounted for by the proportion of non-contractile tissue, but this clearly cannot account for 50% of the leg (muscle plus bone) volume since correction has already been made for the subcutaneous fat, and bone accounts for only about 11% of muscle plus bone volume in the leg (Davies & Sargeant, 1975c).

More importantly, immobilization of the knee in a long leg plaster may produce greater atrophy of the quadriceps muscle than of the other thigh muscles. Also the deep muscle site of biopsy in these patients may not be typical of the whole of the quadriceps under these conditions, although the few available studies (Johnson, Polgar, Weightman & Appleton, 1973; Harris, Hultman & Nordesjö, 1974) suggest that the normal quadriceps in man are relatively homogeneous both structurally and functionally.

A 17% reduction of the $\dot{V}O_{2\ max.}$ achieved in one-leg exercise with the injured leg is associated with the change in leg volume. This again contrasts with the greater reduction in mean fibre area, suggesting that the functional effect of immobilization may be obscured by metabolic activity of other muscles or other regions of the quadriceps in one-leg cycling. However, in a similar group of patients performing one-leg cycling the pattern of forces applied to the cranks with the injured leg was the same as with the uninjured leg (A. J. Sargeant & C. T. M. Davies, unpublished work). This argues against the view that there is markedly greater atrophy (as indicated by the mean fibre area) in the quadriceps as compared with other leg muscles, since it would be surprising if substitution of the hamstrings or gluteal muscles for the quadriceps did not affect the pattern of force application in cycling. Further studies are needed to study any regional

differences in structure and function within the quadriceps, as well as between these and other muscle groups after immobilization.

In conclusion, there is a large degree of fibre atrophy in the quadriceps femoris resulting from disuse after immobilization, and this affects both fibre types. Atrophy is greater than gross measurements of leg volume or function would suggest, possibly because there is relatively less atrophy either in the other thigh muscles, or in other regions of the quadriceps femoris. The nature and functional implications of this differential atrophy require further investigation.

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Forces applied to cranks of a bicycle ergometer during one- and two-leg cycling

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SARGEANT, A. J., AND C. T. M. DAVIES. *Forces applied to cranks of a bicycle ergometer during one- and two-leg cycling.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 42(4): 514-518, 1977. – An examination was made of the comparability of one- and two-leg exercise performed pedaling a stationary bicycle ergometer. The pattern of force exerted on both cranks was examined by means of a specially adapted ergometer which is described. The mean of the peak force in each cycle (MPF) was linearly related to work load ($\dot{W}$) in both forms of exercise, and if account was taken of the doubled work output in two-leg cycling there was no significant difference between the MPF/$\dot{W}$ relationships; these are given by the equations

one-leg: MPF (kg) = 11.23 + 0.065 ($\dot{W}$ in kpm/min)

two-leg MPF (kg) = 10.76 + 0.032 ($\dot{W}$ in kpm/min)

Calculation from the force records of the work performed on the cranks ($W_{cr\ net}$) showed good agreement ($r = 0.98$, $P < 0.001$) with the work load set on the ergometer. Analysis of the proportion of work done in leg extension and flexion phases of cycling revealed no differences between one- and two-leg exercise or between the right and left legs. The majority (~80%) of $\dot{W}_{cr\ net}$ being performed in leg extension is described by: $\dot{W}_{cr\ net}$ (extension) = 10.6 + 0.8 ($\dot{W}_{cr\ net}$ total). In one-leg exercise ($\dot{W}$ > 900 kpm/min) the variation in rotation speed during a normal cycle ranged from +20 to −30% of the mean speed compared with ± 10% in two-leg exercise.

exercise; muscle

IN A NUMBER OF PAPERS, one-leg exercise pedaling a stationary bicycle ergometer has been used to study the physiological responses to exercise in normal subjects and patients (see e.g., 2, 6, 7, 9, 10, 15). However, when consideration is given to the widespread use of stationary bicycle ergometers in physiological and clinical studies, there have been surprisingly few studies of the biomechanical events in normal two-leg cycling and none at all in one-leg cycling. Nevertheless it has often been tacitly assumed that the activities of one- and two-leg pedaling are comparable in that they involve approximately the same movements with the same muscle groups used in the same pattern, although there has been no real evidence to support this assumption. This paper examines and compares one- and two-leg cycling performed by healthy young adults in terms of the pattern of force applied and work performed on the cranks of a stationary bicycle ergometer.

SUBJECTS

The subjects were four healthy young males whose mean ± SD age, weight, and height were, respectively, 26 ± 7 yr, 73.2 ± 12.9 kg, and 176.4 ± 6 cm. The mean maximum oxygen uptakes achieved by the subjects in two-leg and one-leg cycling (5) were 3.39 and 2.48 l/min, respectively. Although all of the subjects could cycle, none of them had taken part in competitive cycling and none had cycled on a regular basis for a number of years. The subjects were, however, allowed unstructured practice of one- and two-leg cycling on the ergometer before the collection of definitive measurements in order to overcome any initial habituation effect (6).

METHODS

The subjects were required to pedal at 50 rpm Van Dobeln-type bicycle ergometer (Signwall) with each leg separately and both legs together in a continuous progressive exercise test. The test was aimed to span the range of the subject's work capacity in four or five work loads lasting 5 min each. The bicycle was a fixed-wheel type and the feet were fitted with plimsolls which were secured to the pedals by means of bolts and two metal plates. Oxygen uptake ($\dot{V}o_2$) and cardiac frequency (f_H) were measured by a continuous open-circuit technique during the last 2 min of each submaximal load and at maximal work loads by the Douglas bag method (6).

The force exerted on the right and left cranks of the bicycle were measured simultaneously and separately by a system based on one described by Hoes et al. (14). Silicon strain gauges were bonded to flat ground surfaces approximately halfway along the trailing and leading edges of both cranks. The input and output to these was effected by mounting discs containing brass slip rings on the bottom bracket spindle inside and fixed to each crank. Spring metal strips were used as pickups in contact with these rings and these were mounted on either side of the bicycle frame just behind the bottom bracket. The output from either crank was fed to separate Wheatstone bridges and then to an ultraviolet oscillograph recorder. Indication of the relative position of the cranks was obtained on the same record by mounting a small photoelectric transistor and lamp either side of the rim of the slip ring disc in which a series of holes had been drilled at 15° intervals and linking the output

to the recorder. The system was calibrated statically by hanging weights from the pedals. Over a period of 6-wk use there was no significant change in the response characteristics of the system, and the coefficient of variation of 20 calibrations made during that period was <2%.

The force measurements were analyzed in two ways. In the first the peak force generated on a crank during each revolution was measured over the last 2 min of each work load and a mean value taken; this is referred to as the mean peak force (MPF). In the second analysis, the work performed on the crank ($\dot{W}_{cr}$) was calculated in a typical revolution by integration of the area between the force record and the zero base line (Fig. 1). The force was measured at each 15° marker position (these are not equidistant due to changes in the speed of rotation during a normal revolution), and the area between marker positions was calculated assuming a mean force and constant speed. The revolution measured was selected from a fast paper speed recording of 10 complete revolutions taken at the end of each work load. The one closest in peak force to the mean measured over the previous 2 min of recording was used. $\dot{W}_{cr}$ was calculated separately for the first 180° of each revolution (starting at top dead center) and the second 180°, these sections corresponding approximately to the leg extension and flexion phases, respectively, of the cycling movement. Throughout the text, "work" is standardized per unit time (expressed as a rate in kilopond meters per minute). Net values of $\dot{V}o_2$ and work have been calculated in all cases by subtracting the zero load value (13, 17).

RESULTS

$\dot{V}o_2$ was consistently higher for a given $\dot{W}$ in one- compared with two-leg exercise, indicating a reduction in apparent mechanical efficiency in one- compared with two-leg exercise (Fig. 2).

Peak force. The peak force of each cycle was measured

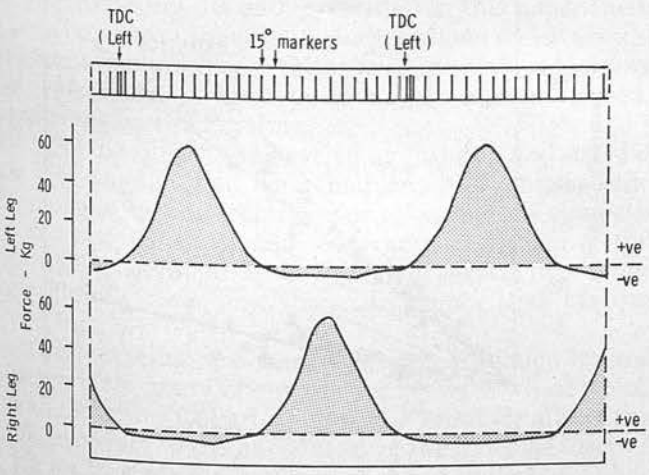

FIG. 1. Force recording at fast paper speed to permit calculation of work performed. Timer marks have been omitted and areas measured to give work performed (positive and negative) have been shaded. The 15° interval markers appear along the top of the record. Left-hand crank top dead center (TDC) is given by a triple marker. Two-leg pedaling is at 1,500 kpm/min.

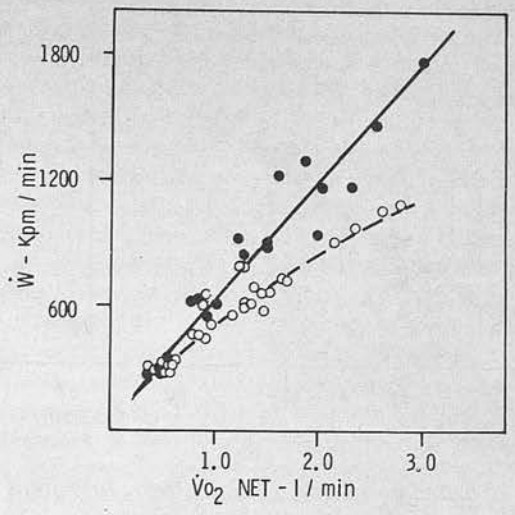

FIG. 2. Work output ($\dot{W}$) in relation to oxygen uptake ($\dot{V}o_2$) in one-leg (○- - -○) and two-leg (●——●) cycling.

over the last 2 min of each work load: the coefficient of variation (CV) of these measurements for a given subject, leg, and work load was ~7%. The coefficient of variation of MPF for the first minute of measurement compared with the second for all subjects at submaximal work loads was <4%.

There was no significant difference between the right and left legs in the MPF applied for given $\dot{W}$ in one-leg exercise for a given subject (Fig. 3). In two-leg exercise, however, there was a consistent and significant ($P < 0.01$: paired t) tendency for the right leg to exert a slightly greater (~3%) peak force than the left.

The relationship of MPF and $\dot{W}$ was linear (Fig. 3) in both one- and two-leg exercise and is given by the following equations

$$\text{one-leg: MPF} = 11.23 + 0.065\ (\dot{W});\ r = 0.98,\ \text{CV} = 12\%$$

$$\text{two-leg: MPF} = 10.76 + 0.032\ (\dot{W});\ r = 0.98,\ \text{CV} = 10\%$$

If account is taken of the doubled work output in two-leg cycling, there is no significant difference between the MPF/$\dot{W}$ relationships in one- and two-leg work, although in both cases there are intersubject differences reflecting slight variations in the pattern of force exerted in cycling.

Work performed on the cranks ($\dot{W}_{cr}$). Preliminary analysis of both one- and two-leg pedaling showed that during the first 180° of the cycle (from top dead center), positive force was applied to the crank and that during the second 180°, a negative force was applied in all but the highest work loads (Table 1, Fig. 1).

$\dot{W}_{cr}$ (positive or negative) from these two phases, which coincide approximately with leg extension and flexion, were therefore measured separately and then added to give a total value. The net total work ($\dot{W}_{cr\ net}$) was obtained by subtracting the work performed pedaling at zero load (see METHODS). Net total work was highly correlated ($r = 0.98$, $P < 0.001$, $n = 48$) with the work load ($\dot{W}$) set on the bicycle ergometer during both one- and two-leg cycling (Fig. 4). Total $\dot{W}_{cr\ net}$ is on average ~5% higher than $\dot{W}$ and the regression relationship for the combined data is given by the equation

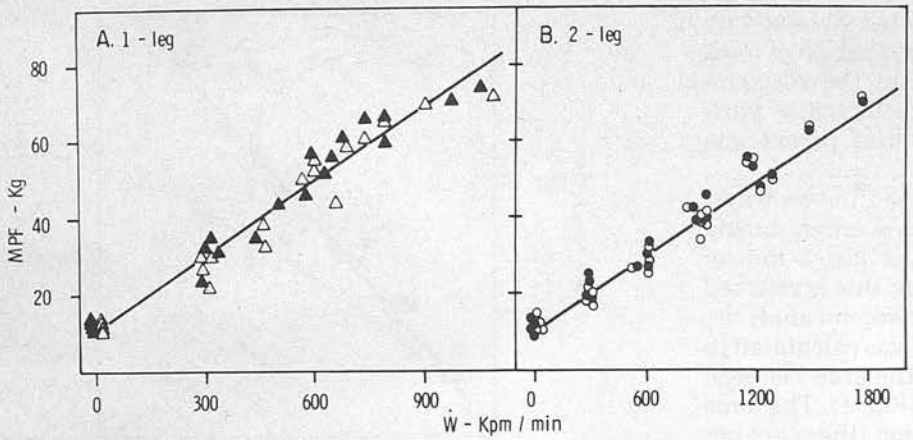

FIG. 3. Mean peak force (MPF) exerted at varying work loads (Ẇ) in *a*) one-leg and *b*) two-leg cycling. One-leg cycling: right ▲; left △. Two-leg cycling: right ●; left ○.

TABLE 1. *Extension (ext), flexion (flex), and total work ($\dot{W}_{cr}$) calculated from the force records*

One-Leg Cycling

Left leg				Right leg			
Ẇ, kpm/min	$\dot{W}_{cr}$, kpm/min			Ẇ, kpm/min	$\dot{W}_{cr}$, kpm/min		
	Ext	Flex	Total		Ext	Flex	Total
0	192	−120	72	0	187	−138	50
	±20	±39	±28		±21	±32	±14
300	440	−73	368	308	454	−88	367
±11	±42	±52	±21	±18	±73	±75	±57
452	574	−54	521	467	603	−71	532
±8	±44	±44	±45	±30	±61	±47	±57
606	709	−24	685	609	732	−33	698
±40	±89	±46	±55	±33	±104	±43	±74
868*	923	+54	977	872*	938	+74	1,012
±172	±180	±54	±232	±170	±184	±71	±248

Two-Leg Cycling

Ẇ, kpm/min	$\dot{W}_{cr}$, kpm/min					
	Left leg			Right leg		
	Ext	Flex	Total	Ext	Flex	Total
0	178	−138	39	188	−147	40
	±39	±25	±17	±30	±33	±33
297	294	−88	205	307	−126	182
±8	±21	±14	±27	±17	±38	±29
597	414	−86	327	432	111	320
±38	±21	±37	±42	±28	±54	±73
888	574	−40	534	582	−81	501
±31	±39	±33	±50	±31	±35	±54
1,184*	729	−37	691	733	−46	687
±37	±17	±33	±51	±24	±11	±29
1,515*	853	+1	854	870	+29	900
±235	±89	±16	±104	±54	±47	±102

Mean ±SD values for the four subjects are given over the range of work loads (Ẇ) set on the bicycle ergometer. *n = 3.

$$\dot{W}_{cr} = 1.127\ \dot{W} - 51.43 \qquad (CV = 9\%)$$

The relative contribution of the extension and flexion phases of cycling to the total $\dot{W}_{cr\ net}$ is shown in Fig. 5. There are no significant differences in this relationship either between legs or between one- and two-leg exercise. Under all conditions and work loads studied ~80% of $\dot{W}_{cr\ net}$ was performed in the leg extension and ~20% in leg flexion phase of cycling. The relationships are given by the equations

$$\dot{W}_{cr\ net}\ (\text{extension}) = 10.6 + 0.8\ (\text{total}\ \dot{W}_{cr\ net})$$

$$\dot{W}_{cr\ net}\ (\text{flexion}) = -10.5 + 0.2\ (\text{total}\ \dot{W}_{cr\ net})$$

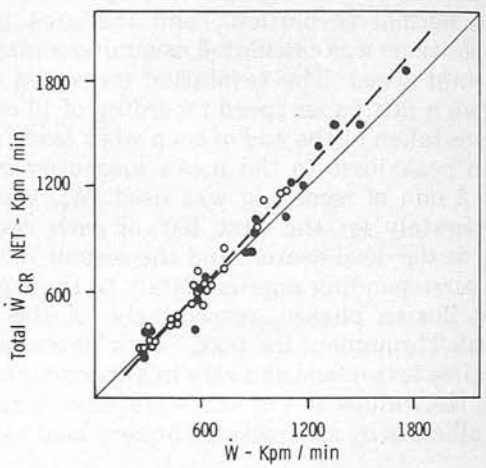

FIG. 4. Relationship of total net work performed on cranks (total $\dot{W}_{cr\ net}$) compared with work load set on the ergometer wheel (Ẇ). One-leg ○; two-leg ●.

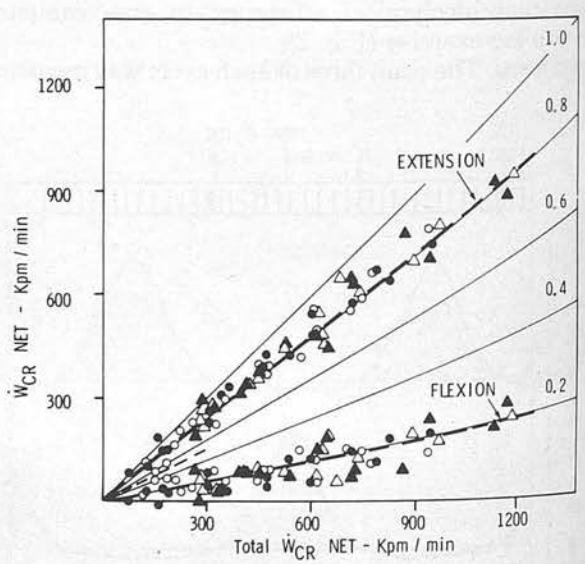

FIG. 5. Relative contribution to total net work performed on each crank (total $\dot{W}_{cr\ net}$) of the extension (top dead center to 180°) and flexion (180° to top dead center), phases of cycling. One-leg exercise: right ▲; left △. Two-leg exercise: right ●; left ○. Regression lines are given (see text) against a background indicating the proportional contribution.

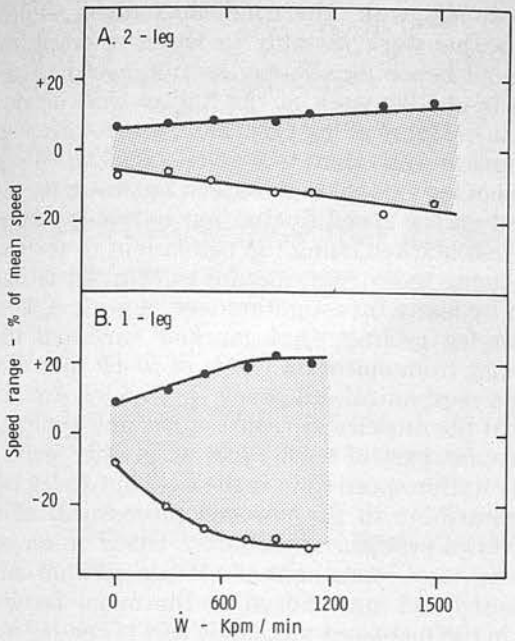

FIG. 6. Maximum (●) and minimum (○) speeds (mean over 15°) of crank rotation expressed as percentage of mean speed for varying work loads during one and two-leg exercise. (*Subject D.*)

Crank rotation speed. In one leg work, deceleration occurs over at least half the cycle whereas in two-leg work this effect is mitigated by the alternating leg action.

Figure 6 illustrates the variation in speed in one- compared with two-leg exercise over a range of work loads for one subject. At 900 kpm/min the effect is such that while the variation in two-leg exercise is approximately ±10% of the mean speed (50 rpm), in one-leg exercise this increases to +20% and −30% of the mean speed.

DISCUSSION

In considering the data presented in this paper there are a number of important qualifications which should be borne in mind. These relate to *a*) the state of training of the subjects, *b*) the type of bicycle ergometer used, and *c*) the speed of cycling.

All of the subjects had cycled as children and still did so occasionally, but none had received any specific training in cycling technique or taken part in competitive sport. Thus although they may be typical of the "normal" population, their pattern of cycling may differ significantly from that shown by competitive cyclists (see, e.g., 14).

One-leg cycling was measured using a friction-braked bicycle of the Van Dobeln type with a fixed wheel which with the active foot attached to the pedal (5) allows the subject to rely on the momentum of the flywheel to carry the leg through the inactive phase of the cycle. Other investigators, however, have adopted different strategies. Gleser (12), for example, had two subjects standing at either side of one bicycle sharing the work between them, and Freyschuss and Strandell (10) used an ergometer which free wheeled and found it necessary to use a spring mechanism to return the pedal to the top of the cycle in order to ensure a smooth cycling movement. Clearly, there may be important differences between these approaches.

All of the experiments reported here were carried out at a nominal pedal speed of 50 rpm in both one- and two-leg exercise, and large variations in this speed will certainly modify the efficiency of cycling (1, 8, 11) and will probably influence the pattern of force application.

Measurement of the peak force of each cycle over a full 2-min period (i.e., 100 cycles) was made after 3 min at each work load. This is in contrast to the relatively brief measurement periods, immediately at the start of exercise, used in studies of two-leg cycling by other investigators (4, 14). As a consequence, our subjects had ample opportunity to accustom themselves to the work load and this is reflected in a coefficient of variation (CV) of ~7% for a given subject and work load. This was attributable to cycle-by-cycle variation rather than periodic changes over the measurement period in *a*) the pattern of cycling or *b*) in the case of two-leg pedaling, work-sharing between the two legs, and consequently the CV of the MPF for the first minute of measurement compared with the second was <4%.

There was no significant difference between right and left legs in the MPF/Ẇ relationship in one-leg exercise, since presumably in order to perform a given Ẇ the same force must be applied unless the pattern of application was significantly altered. In two-leg exercise, however, a slightly (~3%) but significantly greater force is exerted by the right leg. In this context it is interesting to note that all subjects identified the right leg as being their strongest and preferred leg when kicking, jumping, or hopping, although the effect of this preference was not reflected by any significant differences in the leg volume or $\dot{V}O_{2\,max}$ measurements (cf. 16). The range over which force can be effectively applied to the cranks in leg extension and flexion will obviously depend on the saddle position relative to the crank (3, 14) as well as the length of leg segments of individual subjects. However, visual inspection of the force records (see Fig. 1) reveals that very little force is being effectively applied to the crank for a few degrees, either at the top or the bottom of the cycle. We have therefore standardized by dividing the total $\dot{W}_{cr}$ between work done in the first and the second 180° of each cycle calculated from top dead center, and we consider that this will accurately reflect, with very little error, work done in leg extension and flexion, respectively. It is perhaps important to emphasize that the measurement system used in this study only records the force which is applied to the cranks and effectively influences their rotation. Force exerted on the pedals (14) may differ from this if it includes for example compression force exerted when the crank is in the vertical position.

Analyzed in this way it becomes clear that over almost the whole range of submaximal work loads, positive work performed on the crank in leg extension is used both to carry out work on the bicycle (Ẇ) and also to lift the leg during the flexion phase (Table 1). Therefore total work ($\dot{W}_{cr}$) which is the sum of the extension (+ve) and flexion (−ve) phases indicates the work done

on the bicycle. At zero load ($\dot{W}$) total $\dot{W}_{cr}$ is ~50 kpm/min reflecting the work necessary to overcome the frictional resistance in the bicycle transmission as well as in the slip ring assemblies. In order to compare the work load ($\dot{W}$) as set with the calculated values $\dot{W}_{cr}$ it is necessary to subtract from the latter value the $\dot{W}_{cr}$ of pedaling at zero load to give $\dot{W}_{cr\ net}$. When comparison is made in this way $\dot{W}$ and $\dot{W}_{cr}$ are highly correlated (r = 0.98), although $\dot{W}_{cr}$ is on average ~5% higher than $\dot{W}$. This may be related to frictional losses increasing with work load due to imposed stresses on the bearings and bicycle transmission assemblies.

When the net work ($\dot{W}_{cr}$) is calculated separately for the extension and flexion phases of cycling it is clear that there is a progressive increase in the lifting of the leg in flexion (one- and two-leg exercise) that is net $\dot{W}_{cr}$ of flexion increases, although it is not until the highest work load (>90% $\dot{V}o_{2\ max}$) that the absolute value of $\dot{W}_{cr}$ in flexion becomes positive and assists the forward rotation of the crank. The proportional contribution of extension and flexion phases to total $\dot{W}_{cr\ net}$ remains constant at ~80 and 20%, respectively, throughout the range of work loads studied in both one- and two-leg cycling (Fig. 5). Thus in this respect one- and two-leg cycling are comparable activities. However, despite this similarity, apparent mechanical efficiency, as indicated by the relationship of $\dot{V}o_2$ to $\dot{W}$ (Fig. 2), is lower in one- compared with two-leg cycling (5, 9, 10).

Although the proportional contribution of extension and flexion phases does not change in one- compared with two-leg work, the conditions under which they perform this work, notably in terms of crank rotation speed and hence muscle contraction speed, clearly do. Hence in one-leg work at the higher work loads (>900 kpm) at the start of leg extension, the rotation speed of the crank is equivalent to a pedal speed of ~35 rpm; by the end of leg extension the speed has risen to ~61 rpm. In contrast the speed fluctuation in two-leg exercise is much less marked being the equivalent to 45 rpm minimum rising to 55 rpm maximum (Fig. 6). It has been shown by many investigators (see, e.g., 1, 8, 11) studying two-leg cycling, that marked variation in pedal frequency from optimum levels of 50–60 rpm results in reduced mechanical efficiency. In one-leg exercise, the fact that the muscles are contracting and applying force at least for part of each cycle at greater extremes of crank rotation speed than is the case in two-leg pedaling may contribute to the reduced mechanical efficiency. However, a previous explanation based on an increase in the postural component of one-leg work (5) cannot be discounted and may indeed be the major factor implicated in the increased metabolic cost of one-leg exercise. Further evidence is needed on this point and particularly in relation to the influence of speed on apparent mechanical efficiency.

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The effect of disuse muscular atrophy on the forces generated in dynamic exercise

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mary

1. Six patients were studied after prolonged mobilization of an injured leg resulting in scle atrophy.

2. The forces exerted by the atrophied and mal legs during continuous dynamic exer- (one- and two-leg cycling) were examined by ecially adapted ergometer.

3. In one-leg cycling the peak force exerted on crank at a given work rate, the net work rate ormed on the crank, and the proportion of t rate performed in leg extension and n phases of the cycle were the same whether trophied or normal limb was used.

4. Despite these similarities there was an plained reduction in efficiency when using trophied leg to perform one-leg cycling.

5. In two-leg cycling the peak force exerted at en work rate by the atrophied leg was ced by about 40% as compared with the al leg, which reflected a similar reduction e contribution of that leg to the total net rate. Possible reasons and implications this disproportionate sharing of work een the normal and atrophied leg are dis- ed.

words: atrophy, cycling, exercise, force, e, rehabilitation.

duction

ficant differences have been shown be- tween the structure and maximal aerobic function of the atrophied and normal legs of patients during rehabilitation after fracture and consequent immobilization of one limb (Davies & Sargeant, 1975; Sargeant, Davies, Edwards, Maunder & Young, 1977b). However, the effects of this observed functional and structural asymmetry on the forces generated during dynamic exercise are unknown. We have therefore instrumented the cranks of a stationary bicycle ergometer in order to measure the forces exerted by the atrophied and normal legs of patients during one- and two-leg exercise. The effect of limb asymmetry on the sharing of work between the atrophied and normal legs during two-leg exercise was examined, and we have compared one-leg exercise using either the atrophied or normal leg.

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Subjects

The subjects were six young servicemen who had fractured one leg (in four cases the leg preferred for kicking and strength-related activities), which had consequently been immobilized in plaster for an average of 145 days (range 103–183 days). They were attending a residential rehabilitation centre (Joint Services Medical Rehabilitation Unit) where the study was carried out with the approval, and under the auspices, of the Army Personnel Research Committee. All were volunteers and gave their free informed consent. They were seen 62 days (range 49–110 days) after removal of the plaster cast, at which time they were full weight-bearing on the injured leg and had good

union at the fracture site, although their muscles were still weak due to atrophy.

Methods

The subjects pedalled at 50 rev./min a Van Dobeln-type friction-braked bicycle ergometer (Signwall Ltd). Starting at zero load each subject performed a continuous progressive exercise test with each leg separately and both legs together. The test spanned the subject's work capacity at four or five work rates each lasting 5 min. The subjects wore plimsolls, which were attached to the pedals by means of two metal plates and bolts situated under the ball of the foot. Since the bicycle had a fixed wheel this arrangement ensured a smooth action in one-leg cycling, when the momentum of the flywheel carried the exercising leg through the inactive phase of the cycle.

Oxygen uptake was measured by an open-circuit technique and cardiac frequency recorded during the last 2 min of each submaximal work rate, and during the final minute of maximal work rate as previously described (Davies & Sargeant, 1975).

The forces exerted on both cranks of the bicycle were measured simultaneously and separately by strain gauges bonded to flat-ground surfaces on the trailing and leading edges of each crank (Hoes, Binkhorst, Smeekes-Kuyl & Vissers, 1968). Continuous input and output was effected via a slip-ring assembly mounted on the bottom bracket spindle, inside and fixed to each crank. The force data were recorded on an ultraviolet oscillograph along with a marker triggered at every 15° rotation of the cranks. Calibrations made by hanging weights from the pedals showed no significant change over a 6 weeks period (coefficient of variation $<2\%$, $n = 20$; Sargeant Charters, Davies & Reeves, 1977a).

The peak force generated on a crank during each revolution was measured over the last 2 min at each work rate, the mean value being referred to as the mean peak force. The rate of work performed on the crank ($\dot{W}_{CR}$) was also calculated by integration of force and time throughout a typical revolution (selected for being closest in peak force to the mean peak force measured over 2 min). $\dot{W}_{CR}$ was also calculated separately for the first 180° of each revolution (starting at top dead centre) and the second 180°, these sections corresponding to the leg extension and flexion phases of the cycling movement.

Net values of oxygen uptake and work were calculated by subtracting the value obtained when the subject was pedalling against zero load (Hill, 1965; Whipp & Wasserman, 1968).

Leg muscle (plus bone) volume was assessed by an anthropometric technique (Jones & Pearson, 1969).

Values are given as mean ± SD.

Results

Table 1 shows the physical characteristics and maximal exercise responses for the six patients. The average muscle (plus bone) volume of the injured leg was 11% smaller than the uninjured, and this was associated with a similar reduction in the maximum oxygen uptake achieved in one-leg cycling.

Work rate ($\dot{W}$) for a given net oxygen uptake was consistently higher in two- compared with one-leg exercise and higher in one-leg exercise with the uninjured compared with the injured leg (Fig. 1).

TABLE 1. *Age, weight, height, leg muscle (plus bone) volume and maximum oxygen uptake ($\dot{V}O_2$ max.) achieved in exercise with the injured (i) or uninjured (ui) leg and both legs together (two legs)*

Mean values ± SD are shown. $n = 6$ patients.

Age (years)	Weight (kg)	Height (cm)	Leg volume (litre)		$\dot{V}O_2$ max. (l/min)		
			i	ui	i	ui	Two legs
21·8 ±4·4	73·1 ±13·6	174·4 ±5·5	5·73 ±0·87	6·42 ±0·57	2·13 ±0·19	2·42 ±0·30	2·77 ±0·22

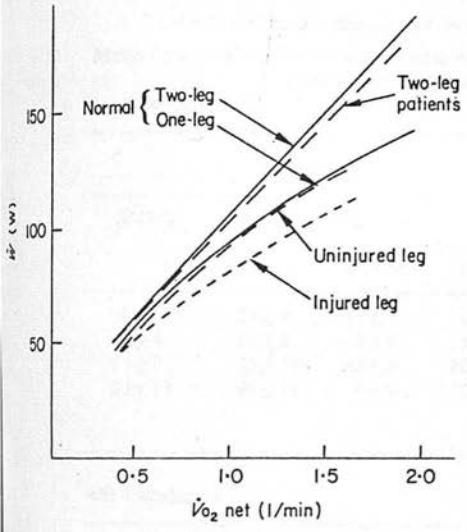

FIG. 1. Work rate ($\dot{W}$) in relation to oxygen uptake ($\dot{V}_{O_2}$) for one- and two-leg exercise performed by normal subjects and patients. The normal data are taken from Sargeant & Davies (1977).

Peak force

The peak force of each revolution was measured over the last 2 min at each work rate: the coefficient of variation of these measurements for a given subject, leg and work rate was 5–10%. There was no systematic difference in the mean peak force of the first minute of measurement compared with the second ($r = 0{\cdot}99$, $P < 0{\cdot}001$, $n = 114$).

Measurements of mean peak force on consecutive days in two subjects using both legs over a range of work loads were highly correlated, with no systematic difference between days ($r = 0{\cdot}99$, $P < 0{\cdot}001$, $n = 20$).

The relationship of mean peak force to $\dot{W}$ was not significantly different in one-leg exercise performed with either the injured or the uninjured leg, although the former failed to reach the same maximal values (Fig. 2a).

During two-leg cycling the mean peak force generated at a given work rate was consistently higher in the uninjured than in the injured leg (Fig. 2b).

Work performed on the cranks ($\dot{W}_{CR}$)

The net work rate performed on the crank(s) ($\dot{W}_{CR\ net}$) was highly correlated ($r = 0{\cdot}97$, $P < 0{\cdot}001$, $n = 95$) in both one- and two-leg cycling with the work rate ($\dot{W}$) set on the bicycle (Table 2). However, in two-leg exercise at a given $\dot{W}$ there was a large and significant difference ($P < 0{\cdot}001$) between the proportion of $\dot{W}_{CR\ net}$ contributed by the injured (38% of the total) compared with the uninjured leg (62% of the total, Fig. 3).

$\dot{W}_{CR}$ of extension and flexion

The relative contribution of the extension and flexion phases of cycling to the 'total' $\dot{W}_{CR\ net}$ performed by a given leg (Fig. 4) shows no significant difference between the injured and uninjured legs in either one-leg or two-leg cycling. The greater proportion of the total $\dot{W}_{CR\ net}$ is performed in leg extension (~75%), whereas only ~25% is performed in leg flexion.

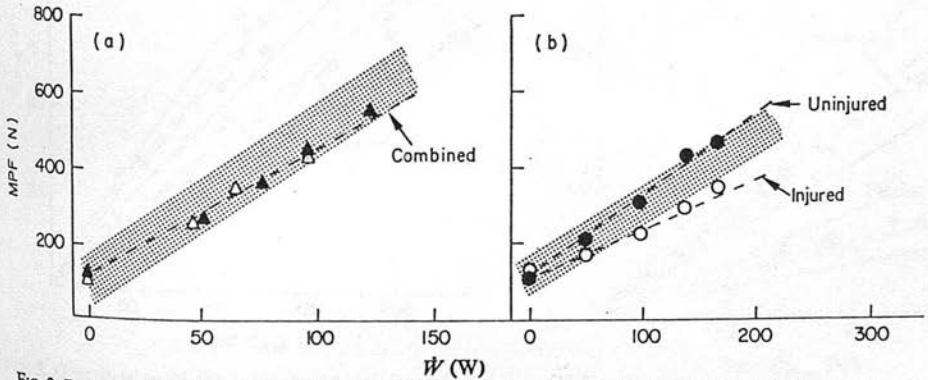

FIG. 2. Relationship of mean peak force (MPF) and work rate ($\dot{W}$). (a) In one-leg exercise, combining data for the injured (△) and the uninjured (▲) leg: MPF = 111+3·36$\dot{W}$, $r = 0{\cdot}97$, SE$\bar{y}$ = 38·2). (b) In two-leg exercise: injured leg (○), MPF = 108+1·29$\dot{W}$, $r = 0{\cdot}92$, SE$\bar{y}$ = 38·4; uninjured leg (●), MPF = 115+2·12$\dot{W}$, $r = 0{\cdot}95$, SE$\bar{y}$ = 44·1). Regressions are based on full data but only mean values at each work load are shown. The shaded area represents the 95% confidence limits of the relationship found in normal subjects (Sargeant & Davies, 1977).

TABLE 2. *Extension, flexion and total work rate ($\dot{W}_{CR}$) calculated from the force records*

Mean (± SD) values for the six patients are given over a range of work loads ($\dot{W}$) set on the bicycle ergometer in (a) one-leg and (b) two-leg cycling.

(a) One-leg cycling

Injured leg				Uninjured leg			
$\dot{W}$ (W)	$\dot{W}_{CR}$ (W)			$\dot{W}$ (W)	$\dot{W}_{CR}$ (W)		
	Extension	Flexion	Total		Extension	Flexion	Total
0	29±7	−19±3	10±7	0	33±5	−18±8	15±7
45±4	60±8	−5±9	55±12	50±2	66±8	−2±6	64±4
64±7	75±14	5±6	80±15	74±5	86±8	2±6	88±7
93±17	96±21	6±8	102±25	94±4	106±10	7±6	112±7
				120±11	122±3	15±3	138±3

(b) Two-leg cycling

$\dot{W}$ (W)	$\dot{W}_{CR}$ (W)					
	Injured leg			Uninjured leg		
	Extension	Flexion	Total	Extension	Flexion	Total
0	31±9	−25±8	7±8	32±10	−26±4	6±7
49±4	43±10	−17±6	26±9	53±7	−17±4	36±5
96±9	60±14	−11±7	49±13	79±14	−10±6	69±14
132±17	67±13	−7±6	60±14	94±13	−3±5	91±14
158±18	74±20	−4±5	70±21	107±11	1±8	108±14

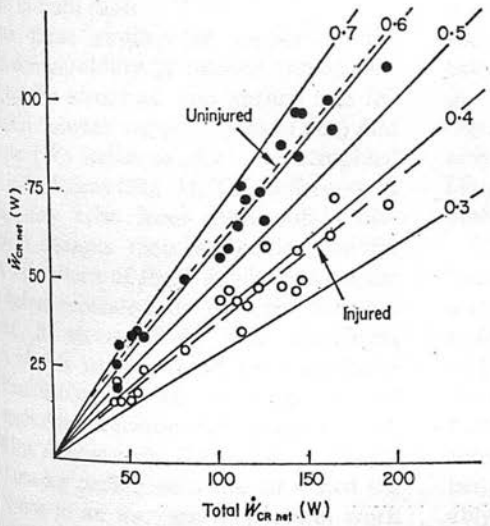

FIG. 3. Contribution of the injured and uninjured legs to the total net work rate (Total $\dot{W}_{CR\ net}$) during two-leg cycling. Individual data points are shown. The regression lines are given by: $\dot{W}_{CR\ net}$ (injured) = 0·39 (total $\dot{W}_{CR\ net}$) − 1·03 (r = 0·96; SE$\bar{y}$ = 5·3), and $\dot{W}_{CR\ net}$ (uninjured) = 0·62 (total $\dot{W}_{CR\ net}$) + 1·03 (r = 0·98; SE$\bar{y}$ = 5·3).

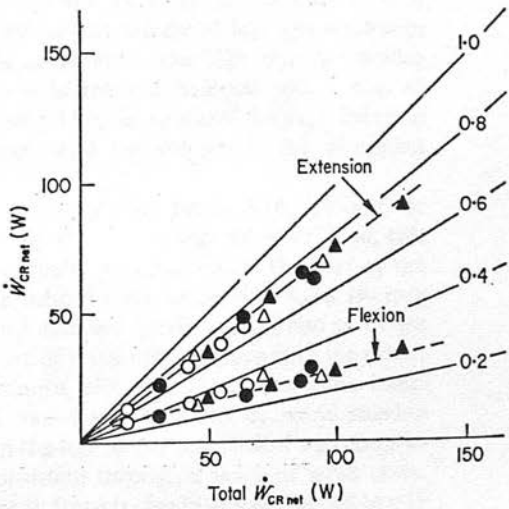

FIG. 4. Net work rate performed on the crank in the extension and flexion phases of cycling. The symbols are used as described in Fig. 2. The regression lines are given by: $\dot{W}_{CR\ net}$ (extension) = 0·76 (total) − 2·6 (r = 0·76; SE$\bar{y}$ = 5·8), and $\dot{W}_{CR\ net}$ (flexion) = 0·24 (total + 2·6 (r = 0·76; SE$\bar{y}$ = 5·8).

Discussion

Although in previous studies in both patients and normal subjects it has been tacitly assumed that one- and two-leg cycling, and one-leg cycling with an affected and unaffected limb, are comparable activities involving the same movement pattern, this has never been established (Dunér, 1959; Carlson & Pernow, 1959; Freyschuss & Strandell, 1968; Gleser, 1973; Davies & Sargeant, 1975; Saltin & Landin, 1975; Saltin, Nazar, Costill, Stein, Jansson, Larsen & Gollnick, 1976).

One-leg cycling

We found no significant difference during one-leg exercise between the atrophied (injured) and normal (uninjured) legs in the relationship of mean peak force to work rate although the peak rate achieved by the atrophied leg was lower (Fig. 2a). There was also no significant difference in the relationship between net work rate performed on the cranks ($\dot{W}_{CR\ net}$) to the rate set on the ergometer (Table 2) for the two legs. When the pattern of work was characterized by the relative contribution of extension and flexion phases in one-leg cycling, no differences were found between atrophied and normal legs (Fig. 4), the largest proportion (>75%) of $\dot{W}_{CR\ net}$ being generated by leg extension in both cases.

Despite these similarities supporting the assumed comparability of one-leg cycling performed by the atrophied and normal legs the relationship between oxygen uptake ($\dot{V}O_2$) and work rate ($\dot{W}$) indicates that the atrophied leg was less efficient (Fig. 1). This difference in efficiency may arise from more subtle biomechanical changes than we could identify, such as the pattern of force application on the cranks being generated by different muscles. However, it seems likely that significant changes of this nature would have produced some identifiable variation in the pattern of force application between the atrophied and normal leg. Alternatively, the increased oxygen cost of one-leg cycling with the atrophied leg may be due to an increase in postural work (Davies & Sargeant, 1975), although it is difficult to see why the same work rate should require an increased postural effort at the low rate at which the $\dot{W}/\dot{V}O_2$ relationships of the two legs begin to diverge. Yet again, the difference may reflect variations in the metabolic efficiency of the atrophied and normal muscle, dependent upon the velocity of contraction and the relative contribution of type I or type II muscle fibres (Goldspink, Larson & Davies, 1970; Bolstad & Ersland, 1976), as more and possibly metabolically less-efficient fibres for the prevailing conditions will need to be recruited in the atrophied muscle in order to generate the same force.

Two-leg cycling

In two-leg cycling the relationship of mean peak force to work rate is significantly different for each leg (Fig. 2b). In net terms mean peak force is about 40% lower in the atrophied compared with the normal leg for a given work rate. This striking difference is also reflected in the $\dot{W}_{CR\ net}$ calculated separately for each leg so that the atrophied leg is contributing about 40% less work towards the total than the normal leg (Fig. 3). However, the proportional contribution to $\dot{W}_{CR\ net}$ of extension and flexion remains the same for each leg and is not significantly different from that found in one-leg exercise (Fig. 4).

Thus it appears that the 'pattern' of cycling with the atrophied leg, whether used in one- or two-leg exercise, remains the same as with the normal leg, but in two-leg exercise the atrophied leg operates at a work rate which is about 40% below that of the uninjured leg. The difference in $\dot{W}_{CR\ net}$ between the legs during two-leg exercise is in marked contrast with a loss of only about 11% in maximal aerobic function ($\dot{V}O_2$ max.) and leg volume in the atrophied limb.

This anomaly may result from 'resting' or 'protecting' the injured leg. However, if so, this was apparently unconscious on the part of the patients, who did not know that work sharing was being assessed during the exercise and were not aware of using one leg more than the other. Furthermore, although there was some inter-subject variation, the ratio of work sharing between the legs in any one patient was remarkably consistent through a range of work rates. Mean peak force is also highly correlated in any one patient and leg during two-leg cycling whether examined on a test/re-test basis over a wide range of work loads or on a minute-to-minute basis (coefficients of variation <4%).

The consistency of these latter findings suggests that there may be a physiological basis for the disproportionate work sharing between the legs, although it is not clear what this may be. One possibility may be that the ratio of work sharing is related to optimal efficiency for the same number and type of motor units representing the optimum for efficiency under the prevailing conditions (Goldspink *et al.*, 1970; Bolstad & Ersland, 1976), are recruited in both legs but that those in the trained leg produce 40% less tension as a result of atrophy. In support of this view it should be noted that biopsies from the quadriceps muscle of amputee patients show a reduction of about 40% in cross-sectional fibre area, whereas limb present patients they showed a reduction in $\dot{V}O_2$ max. and gross measurement of leg volume of only about 12% (Sargeant *et al.*, 1977a).

Whether disproportionate work sharing in two-leg cycling is simply due to 'protecting' the atrophied leg or whether it reflects a real physiological difference it has important implications. Two-leg cycling, and possibly other clinical exercises, may not produce the maximal desired effect when used as part of a practical programme of rehabilitation unless carefully monitored. It also suggests that after immobilization there is a considerable physiological difference between the limbs which is not known by measurement of maximal oxygen uptake or leg volume, but which may reflect changes occurring at a cellular level in the atrophied muscle.

Acknowledgments

We are indebted to the staff and patients of the Joint Services Medical Rehabilitation Unit, R.A.F. Chessington, without whose enthusiastic and willing co-operation this study would not have been possible. We also thank Mr A. Charters and Mr E. Reeves for constructing the equipment used in this study, and Mr K. Jones for assistance in the analysis of the data.

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FUNCTION AND DIMENSIONS OF THE CIRCULATORY SYSTEM IN ANOREXIA NERVOSA

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ABSTRACT. Fohlin, L., Freyschuss, U., Bjarke, B., Davies, C. T. M. and Thorén, C. (Department of Paediatrics, Karolinska Institutet, St. Göran's Hospital, Stockholm, Sweden). Function and dimensions of the circulatory system in anorexia nervosa. Acta Paediatr Scand, 67: 11, 1978.—The functional and dimensional components of the oxygen transporting system was studied in 17 female and 11 male patients suffering from anorexia nervosa. Both groups were 14.9 years old, on average, and had lost about 25 % of their weight. Measurements at rest included blood and heart volume, heart rate, blood pressure, oxygen uptake ($\dot{V}O_2$), RQ, blood lactate (LA) and in 6 of the patients cardiac output. During bicycle ergometry the determinations of heart rate, blood pressure, LA, $\dot{V}O_2$ and cardiac output were repeated and maximal aerobic power was determined. A low metabolic rate with bradycardia and hypotension was apparent at rest. Blood and heart volume was decreased proportionally to the weight loss. On a given work load $\dot{V}O_2$ was lowered to the same extent as the resting metabolic rate. At maximal effort $\dot{V}O_2$ was reduced out of proportion to the circulatory dimensions and maximal heart rate was low. During exercise cardiac output was normally related to $\dot{V}O_2$ and stroke volume was maintained, indicating a normokinetic circulation and an unimpaired myocardial function. The main cause of the low maximal aerobic power seems to be the reduced muscle mass.

KEY WORDS: Blood volume, blood lactate, cardiac output, children, heart volume, maximal exercise, oxygen uptake

In anorexia nervosa (AN) symptoms from the cardiovascular system are often apparent such as bradycardia, hypotension and acrocyanosis (8, 28). The presence of unspecific ventricular repolarization changes on the ECG (23, 27) have evoked questions of the myocardial involvement in the disease. Although many patients have a hyperactive behaviour and cardiovascular symptoms are common, there are no data in the literature regarding function of the circulatory system in anorexia nervosa. The potentially very poor prognosis—a mortality rate of about 10 % being reported—and unexpected sudden deaths (6, 21, 24, 28) initiated a series of studies (1, 10, 11, 14–16) to evaluate the cardiovascular accommodation to the long-standing caloric starvation in AN patients. In the present study various dimensional components of the oxygen transporting system have been examined at rest and during submaximal and maximal exercise performance.

MATERIALS AND METHODS

Seventeen female and 11 male patients were studied after the informed consent by the patients as well as by their parents and after the approval by the Ethical Committee of Karolinska Institutet. The patients selected for the investigation all conformed to the following criteria chiefly from Dally (8):

1. age at onset less than 25 years
2. active refusal to eat with accompanying pronounced weight loss

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Table 1. *Physical characteristics of 17 female and 11 male anorexic children*

Individual data and mean values ±S.D. of the girls and boys are given

	Age (yrs)	Weight (kg)	Height (cm)	Per cent weight loss	Duration (yrs)
Girls					
H. H.	12.1	30.0	153	15.0	0.5
G. Z.	12.9	24.5	153	30.0	0.5
C. W.	13.1	39.0	163	35.0	0.5
H. Hj.	13.2	40.5	168	19.3	0.9
E. A.	13.3	33.5	157	14.1	0.5
M. E.	13.6	48.5	169	21.8	0.8
M. St.	14.0	35.0	164	27.1	0.6
C. P.	14.2	34.5	157	14.8	1.3
A. S.	14.8	42.9	173	24.5	1.0
G. G.	15.2	34.0	158	39.8	1.5
K. Å.	16.7	38.0	173	32.0	2.0
A. H.	15.9	35.8	164	33.7	1.5
Y. S.	16.6	48.2	169	25.9	2.0
P. W.	16.7	35.2	166	30.5	1.0
U.N.	16.9	41.8	180	25.9	1.0
L. P.	17.6	42.5	162	24.1	0.6
C. A.	17.7	40.5	163	32.5	3.0
Mean	14.9	37.8	164.2	26.2	1.1
S.D.±	1.8	6.1	7.4	7.5	0.7
Boys					
M. M.	11.3	28.6	142	37.8	2.0
J. W.	12.0	27.3	146	20.4	0.9
L. K.	12.5	31.0	156	20.5	1.0
B. G.	13.7	50.6	188	24.5	1.0
P. W.	14.0	38.5	164	23.0	0.8
T. G.	15.2	36.6	164	22.1	0.5
T.L.	15.9	36.3	164	30.0	1.0
T. T.	16.0	56.7	187	22.2	0.8
M. S.	17.1	51.0	184	22.0	1.0
U. P.	17.5	49.0	184	28.0	1.0
A. N.	18.2	48.7	186	21.5	1.0
Mean	14.8	41.3	169.6	24.7	1.0
S.D.±	2.3	10.3	17.1	5.3	0.4

3. no evidence of schizophrenia, severe depression or organic disease.

The psychiatric condition of patients varied, but none was primarily psychotic.

Amenorrhoea was present in all the postpubertal girls. Four girls had not passed menarche when they started to lose weight and 3 boys had no signs of puberty. Mean age of both the girls and boys was 14.9 years.

The physical characteristics of the AN patients and duration of the weight loss are given in Table 1. The body weight in all except 4 was less than −2 S.D. on the growth chart, relating body weight to height; their average weight loss was about 25%. None had edema or anemia and blood electrolytes were within normal limits. One boy later died due to septicemia following intravenous nutrition.

The investigation was performed while the patients were treated at St. Göran's Children's Hospital. Three patients were on drug therapy (Thioridazin chloride, Mallorol®). The study was completed during the years 1972–76.

The patients were measured in accordance with standard procedures for body weight (kg) using a clinical balance accurate to±0.1 kg and height (cm) using a stadiometer accurate to ±0.5 cm. Blood volume was determined with ^{125}I-labelled albumin (29), plasma volume being derived from blood volume and hematocrit determinations. The heart volume (HV, ml) was measured in the prone position (20). Expiratory gas was collected in Douglas bags and the volumes were measured with a Tissot-spirometer. Gas samples were analysed for oxygen and carbon dioxide by the micro-Scholander technique. Heart rate (HR) was obtained from ECG. Blood lactate (LA) was determined enzymatically (7) from arterialized (finger prick) blood. Cardiac output was determined by the dye dilution technique with indocyanine-green (Cardio-Green®) as the indicator and a Beckman densitometer as recording unit. Resting measurements were taken in supine position. Exercise was performed in the sitting position on an electrically braked bicycle ergometer (Elema) at a pedalling rate of 60 rpm. During exercise HR was measured every minute; minute volume of ventilation and oxygen uptake were determined during the third–sixth minute of the work load. The exercise tests were performed with a stepwise increase of loads, which usually comprised two submaximal and one maximal load. It was not always possible to apply the "levelling off" criteria of $\dot{V}O_2$ max. The patients were simply encouraged to pedal the bicycle to exhaustion. The secondary criteria of blood lactate and respiratory quotient (2, 3) were used as evidence of maximal effort. Seven failed to fulfil these criteria.

RESULTS

Mean values of the circulatory and metabolic data measured at rest before the exercise test in the AN children are summarized in Tables 2 and 3. The blood volume per kg body weight

Table 2. *Circulatory data of the anorexic children obtained at rest*

Mean values ±S.D. are presented. Within parentheses are numbers of patients when fewer than the whole series

	Girls (n=17)	Boys (n=11)
Heart volume, ml	437±74.2	547±135.9 (9)
Hemoglobin, g×l^{-1}	138±12.6	135±10.5
Blood volume, l	2.79±0.47 (14)	3.26±0.82
Blood volume/body weight, ml ×kg^{-1}	75±11.8 (14)	80±13.5

Table 3. *Circulatory and metabolic data of patients with AN at rest and during maximal exercise*
Mean values ±S.D. are given. Within parentheses: numbers of patients when fewer than the whole series

	Rest		Maximal work	
	Girls (n=17)	Boys (n=11)	Girls (n=13)	Boys (n=8)
Load, W			99±13.2	113±25.5
Oxygen uptake, l STPD×min^{-1}	0.139±0.018 (12)	0.153±0.037 (6)	1.21±0.18	1.38±0.33
Oxygen uptake per kg body weight, ml STPD×kg^{-1}			32±5	35±3
R	0.81±0.10 (12)	0.82±0.06 (6)	1.14±0.07	1.18±0.07
Blood lactate, mmol×l^{-1}	1.7±0.9 (11)	1.8±1.2 (7)	11.8±2.2	12.6±2.1
Heart rate, b.p.m.	53±9.3	52±11.7	174±8.2	175±5.7
Blood pressure, mmHg				
syst	97±9.2	96±10.0	145±20.9 (9)	149±17.4 (6)
diast	65±5.2 (15)	67±6.0		
Minute volume of ventilation per l oxygen uptake			41.8±7.08	39.1±6.30

was in both girls, 75 ml, and boys, 80 ml, within the limits of healthy children as determined by the total hemoglobin method (18). For a given body weight heart volume was within the range expected for healthy children (14, 26) except in 5 of the patients (Fig. 1). However, in the relationship the majority of points were above the regression line. In the AN children the relation between blood volume and heart volume was highly significant (r=0.80, p<0.001). The oxygen uptake at rest was on the average about 20% lower than predicted from sex, age and body surface area and both girls and boys showed evidence of bradycardia 53 and 52 b.p.m., and hypotension, 97/65 and 96/67 mmHg respectively. Three patients had shortly before this examination an extreme bradycardia of about 30 beats per minute. Sinus rhythm dominated, but periods of sinus arrest and ectopic atrial rhythm were recorded. The mean values of blood lactate 1.8 mmol ×l^{-1}, were within the upper normal range of the analysing laboratory (26).

During exercise the oxygen uptake for a given work load was lower than expected (Fig. 2); the regression line was displaced parallel to and below the normal relationship (3). $\dot{V}_{O_2\,max}$ averaged 1.21 ×min^{-1} in the girls

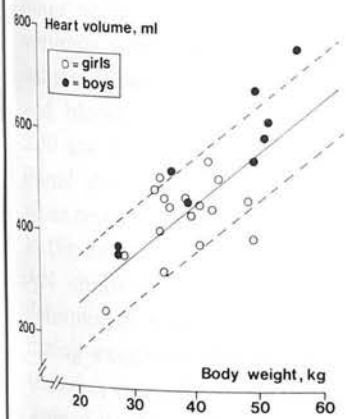

Fig. 1. Heart volume in relation to body weight in 26 children with AN (Y=59.88+10.49x, r=0.78***). The regression line ±2 S.D. of 109 healthy children (Y=61.93+10.06x; r=0.86***) (26).

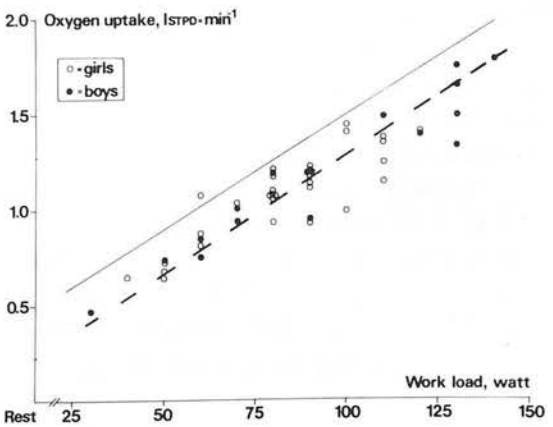

Fig. 2. Oxygen uptake of various work loads in 28 AN patients. --- represents AN (Y=0.222+0.0105x, r=0.85***, n=46);—denotes regr. line of healthy children (3).

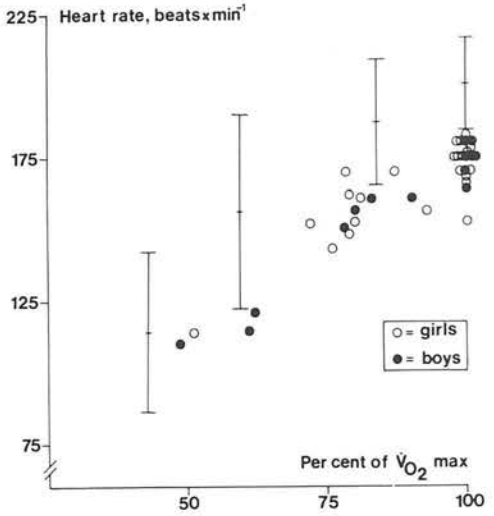

Fig. 3. Heart rate and fraction of measured maximal oxygen uptake in 21 AN children. For comparison corresponding mean values ±2 S.D. of 13–14-year-old boys are given (12).

and 1.38 $l \times min^{-1}$ in the boys (Table 3). In terms of body weight this represented a $\dot{V}O_2$ of 32 $ml \times kg^{-1} \times min^{-1}$ and 35 $ml \times kg^{-1} \times min^{-1}$. These values are outside the lower range of values found in healthy children of the same age (25). The regression line relating $\dot{V}O_{2\,max}$ to heart volume was displaced below the expected relationship (14, 26). The maximal heart rate was 174 $beats \times min^{-1}$ in the girls and 175 $beats \times min^{-1}$ in the boys. The respective maximal lactate values were 11.8 and 12.6 $mmol \times l^{-1}$ and systolic blood pressure averaged 145 and 149 mmHg, respectively. During exercise HR for a given relative $\dot{V}O_2$ was lowered (Fig. 3). The normal relationship between cardiac output and $\dot{V}O_2$ demonstrated in Fig. 4 for 6 patients indicates that the AN children have a normokinetic central circulation when compared with healthy young boys (12). The calculated stroke volumes were well maintained during exercise (Fig. 5).

DISCUSSION

The higher proportion of boys in this study than the usual reported incidence of about 10% (4, 5) is partly due to a more active selection of boys.

The patients in the present study have low heart rate and subnormal blood pressure at rest. These findings are characteristic for long-standing starvation (19) and together with the low oxygen consumption at rest they are all probable phenomena of the adaptation to a low metabolic rate.

Heart and blood volume were found to be normally related to body weight. Hence the decrease in the dimensions of the cardiovascular system seems to be proportional and secondary adapted to the body weight.

During exercise the heart rate increased linearly but the maximal heart rate was significantly lower than normal. Some caution is necessary when interpreting these findings since it was impossible to apply generally accepted levelling off criteria for maximal work in these patients. However, R values of about 1.1, ventilatory equivalent of around 40 and the high blood lactate values following the work test support the view that the patients were at or near their maximal performance. Further studies in AN children before and after rehabilitation with weight gain showed an increase of the maximal heart rate to a normal level (15). From Fig. 3 it appears that also at comparable relative loads the heart rate was lower than in healthy boys (12), but this reduc-

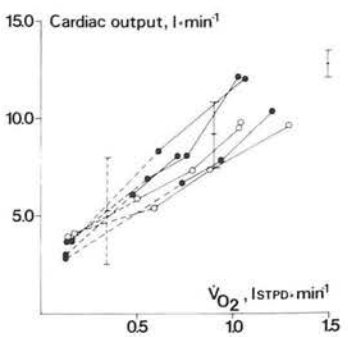

Fig. 4. Cardiac output in relation to oxygen uptake at rest, supine and during upright exercise in 6 AN patients. Corresponding mean values ± 2 S.D. of 13–14-year-old boys are included (12). --- refer to resting conditions. Symbols as in Fig. 1.

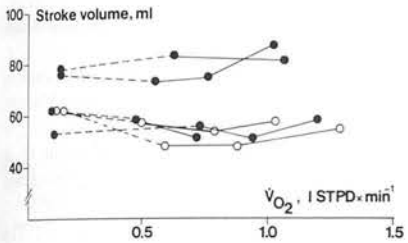

Fig. 5. Stroke volume in relation to oxygen uptake at rest, supine and during upright exercise in 4 AN boys and 2 AN girls. Symbols as in Fig. 1.

tion was of the same order as that seen at rest. A low body temperature and metabolic rate are constant findings in anorexia nervosa and as hypothermia is associated with a low heart rate (9, 22), this may be a factor contributing to the lowered heart frequency observed.

Cardiac output at rest and during work was within 1 S.D. and −2 S.D. of the values found in 13–14-year-old boys (12), and it was normally related to oxyen uptake. Stroke volume was well maintained during work. These findings indicate a normokinetic circulation and an unimpaired myocardial function. At roughly similar levels of oxygen uptake during exercise stroke volumes in 2 of the AN boys were within ±S.D. of the values found in healthy boys (12), while values below the lower normal range was observed in 2 AN boys and 2 AN girls. When similarly compared with another series of healthy boys (13), all stroke volumes were within ±S.D. of the reference values. The relation between stroke volume and blood volume varied between 1.85 and 2.30 and hence similar to corresponding ratios found in healthy adults (17) in turn indicating all an ordinary adaptation of the stroke volume to the circulating blood volume. In 5 of the 6 AN children, in whom cardiac output was determined, ECG was normal both at rest and during exercise; the sixth patient had at one occasion unspecific ST-T changes, which remained unchanged during exercise; within 10 days ECG had normalized.

In 6 other patients T-wave inversions were recorded in the precordial leads during exercise. These changes became, however, less marked with increasing work loads. Further, during a preceding orthostatic test a similar ECG pattern was observed, and it could be abolished by the administration of a β-receptor blocking agent (Inderal®). It therefore seems probable that these repolarization changes were of functional origin.

For a given absolute work load the oxygen uptake was significantly reduced. This might lead one to suspect that AN children pedal with greater mechanical efficiency than normal, but the regression line between oxygen uptake and load reveals a parallel displacement of the same magnitude as their resting metabolic rate.

The major effect of anorexia nervosa on physical performance is undoubtedly a reduction in maximal aerobic capacity with a low maximal heart rate. Absolute maximal oxygen uptake and also its relation to body weight were both low when compared to normal children of same age. The altered association of $\dot{V}O_{2\,max}$ to heart volume indicates that working capacity measured as aerobic power decrease independently and out of proportion of the circulatory dimensions. The main limiting factor should then be the reduced muscle mass and possibly the low maximal heart rate. AN undoubtedly has a debilitating effect on the patient and this may partly account for the reduced aerobic performance during later and advanced stages of the disease.

ACKNOWLEDGEMENT

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TOTAL BODY POTASSIUM FAT FREE WEIGHT AND MAXIMAL AEROBIC POWER IN CHILDREN WITH ANOREXIA NERVOSA

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ABSTRACT. Davies, C. T. M., von Döbeln, W., Fohlin, L., Freyschuss, U. and Thorén, C. (The Department of Paediatrics, Karolinska Institutet at ST. Göran's Children's Hospital and the Aeromedical Laboratory of the Swedish Medical Research Council at Gymnastik- och Idrottshögskolan, Stockholm, Sweden). Total body potassium fat free weight and maximal aerobic power in children with anorexia nervosa. Acta Paediatr Scand, 67: 229, 1978.— Body composition and aerobic work performance have been studied in 5 boys and 10 girls suffering from anorexia nervosa. The average ages of the two groups of children were 15.4 (boys) and 15.2 (girls) years respectively. Measurements of body composition included height, weight (*W*), body potassium (^{40}K), skinfold thickness (SFT) at triceps and subscapularis, blood volume (BV) and femoral condylar and radioulnar breadths. From these measurements estimates of fat free weight (FFW), skeletal weight (*S*) and lean body mass (LBM) were made. Work performance was assessed by measurement of the maximal aerobic power ($\dot{V}_{O_2}$ max).

The patients had lost on average 26 % of their former body weight. The boys had on average >7 % of their body weight as fat compared with >9 % in the girls. However, the loss of weight was not solely due to loss of body fat, but could also be ascribed to a decrease in soft fatfree tissue. LBM or FFW could be estimated as well from SFT as from ^{40}K. $\dot{V}_{O_2}$ max averaged 1.43 l/min (35.1 ml/kg/min) in the anorexic boys and 1.24 l/min (33.2 ml/kg/min) in the girls and was associated with FFW and LBM. However, $\dot{V}_{O_2}$ max was lower in relation to LBM than in healthy children of the same age. Thus it was suggested that the emaciation in anorexia is directly attributable to loss of both fat and muscle and accounts in part for the reduction of aerobic power observed. However, an important factor may be the debilitating effect of starvation on the patient, particularly in its advanced and later stages, which reduces his/her level of habitual physical activity.

KEY WORDS: Anorexia nervosa, body composition, children, exercise, lean body mass, maximal oxygen uptake

There have been several studies of body composition in children (13, 28, 31), young healthy adults (12, 19, 34, 35) and obese patients (6, 15) and the development of the total body potassium (^{40}K) method for estimating fat free weight (FFW) has increased our knowledge of body structure in health (4, 5) and disease (3, 23, 29). The effect of severe caloric deprivation has been documented in civilians and prisoners of war (26), under carefully controlled laboratory conditions in young healthy adults (26), and in patients undergoing restricted diet treatment for obesity (25) and in adult patients with anorexia nervosa (20, 27).

Nevertheless there still remains a lack of information of diet starvation on body structure and physiological function in the pubescent child. In the present investigation

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Table 1. *Body composition, Blood volume and maximal oxygen uptake in anorexia nervosa patients*

Mean ±S.D. Abbreviations see text. Within brackets the number of patients when less than the series

	Age (y.)	Ht (cm)	*W* (kg)	Weight-loss (%)	LBM (kg)	*K* (g)	*S* (kg)
Boys=5	15.4±2.5	169±17	40.8±10.1	23±4	38.0±8.5	81.5±11.8	10.3±2.5
Girls=10	15.2±1.9	163±8	36.1± 6.4	28±7	32.8±4.7	75.7±11.4	8.2±0.9

we have examined body composition, including measurements of ^{40}K in 15 patients suffering from anorexia nervosa and related our findings to maximal aerobic power ($\dot{V}_{O_2}$ max) measured on a bicycle ergometer.

MATERIAL AND METHODS

The material for this investigation was drawn from a series of anorexia nervosa (AN) patients on whom measurements of thermoregulatory function and the dimensions of the O_2 transporting system have been previously reported (14, 21). In the present study, 15 patients (10 girls and 5 boys) were measured on separate occasions. All patients selected for this study conformed to the following criteria formulated previously (21) for AN.

1. Age at onset less than 25 years
2. Active refusal to eat with accompanying pronounced weight loss
3. No evidence of schizophrenia, severe depression or organic disease.

Amenorrhoea was present in all the post-pubescent female patients and none of the patients had edema. The *weight, height* and *skinfold thickness* were measured using a clinical balance accurate to ±100 g, a stadiometer accurate to±1 cm and Harpenden skinfold caliper accurate to ±5% respectively.

From the sum of the two skinfolds, triceps and subscapularis, *lean body mass (LBM)* was calculated according to Pariźková (30). A multi-stage *exercise test* was performed on an electrical braked bicycle ergometer (Siemens-ELEMA) with stepwise increased work loads up to maximal level, i.e. to the limit of their exercise capacity. *Oxygen uptake* was determined from expired air collected successively in Douglas bags during the last minutes of the final work loads. Expired air volume was determined by evacuating the bag through a Tissot spirometer and O_2 and CO_2 contents were measured by the micro-Scholander technique. *Blood lactate* (LA) was determined immediately and 4 min after cessation of exercise from pre-warmed ("arterilialised") finger prick sample after an enzymatic method (10). The criteria for maximal *aerobic power* ($\dot{V}_{O_2}$ max) having been reached were that LA should exceed 9 mM/l and respiratory quotient should exceed unity (2).

Whole body potassium (^{40}K) was determined in a whole body counter on the aeromedical laboratory. The counting of the gamma emission from the body of the subject was performed within a chamber with lead walls, 80 mm thick. The subject was sitting reclined in a standard chair with an angle of 90° between the body and the thigh. In the center line of this angle the center of a plastic scintillator of the size 20×10×6.5 inches was placed. The signals from the photomultipliers attached to the plastic scintillator were analyzed in a 100-channel analyzer. Counting time was 2400 s.

The calibration of the gamma counter had earlier been done using male and female adult subjects whose body composition was known from density determination by hydrostatic weighing. Their potassium content was estimated using the values given by Forbes & Lewis (22). The total error of the method was found to be between 5 and 6%. The statistical error derived from Poisson's law is for subjects with 130 g potassium 2.2%.

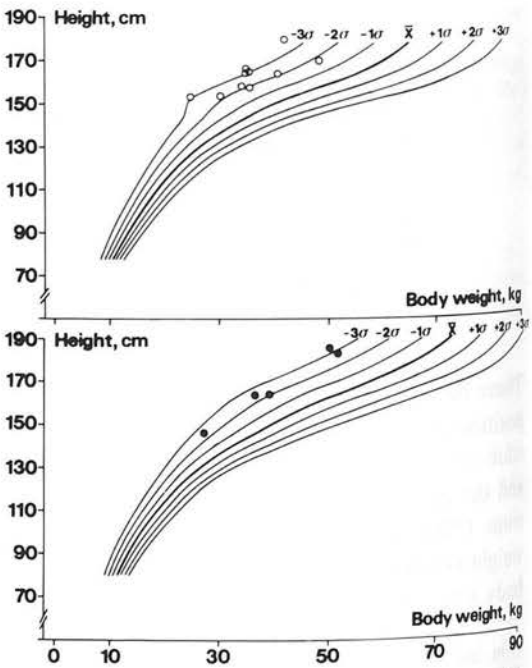

Fig. 1. Relationship of weight to height in anorexic girls and boys. The standard height–weight curves of normal healthy Swedish children are shown. Girls (○), boys (●).

FW (g)	BV (l)	$\dot{V}_{O_2}$ (l/min)
1±6.2	3.16±0.64	1.43±0.32
9±3.9	2.76±0.49	1.24±0.19 (8)

Using the potassium content of the body the weight of the soft fatfree tissue of the test subject was estimated from the formula:

$$ST = \frac{K - 1.1S}{2.7}$$

where ST=soft fatfree tissue (kg), K=g potassium from gamma emission, S=weight of skeleton (kg) from measurements of body height, the sum of right and left condylar breadths and the sum of the right and left radioulnar breadths using the empirical formula developed by von Döbeln (17, 18). The constants represent the normal potassium concentration of the skeleton and the soft fatfree tissue respectively. Fatfree weight (FFW) was derived from the sum of ST and S (17, 18).

RESULTS

The basic data are summarized in Table 1. The patients are approximately −3 S.D. below their expected weight for height (Fig. 1). Calculated from skinfold thickness the boys had 6.4 ±2.5% of their total body weight as fat; the corresponding figure in the girls is 8.5 ±3.5%. Skinfold thickness at triceps and subscapularis average 3.4 and 4.0 mm for the boys and 5.4 and 4.5 mm for the girls. These values are well outside the lower normal range given by Tanner (32) (Fig. 2). In absolute values ^{40}K was reduced compared to normal children of the same age, but in terms of body weight ^{40}K was 2.12 g/kg in the boys and 2.10 g/kg in the girls. There were significant correlations between ^{40}K and body weight with a coefficient (r) of 0.97 and 0.80 for the boys and girls respectively. The regression equation relating ^{40}K to body weight, height and LBM for the male and female patient are given in Table 2. A highly significant relationship of FFW, estimated from ^{40}K and skeletal weight, to LBM, determined from skinfold thickness, was found (Fig. 3). However, by the method of calculation of FFW it was not possible to detect any fat at all in two patients in whom the skinfold thickness gave a result of about 4% fat. Skeletal weight accounted for on average 25% of the body weight in the boys and 23% in the girls. The corresponding mean values for healthy young male and female subjects are 19% and 17% (16). There was also a significant (r=0.97) relationship between $\dot{V}_{O_2}$ max and LBM (Fig. 4).

DISCUSSION

Interest in the field of human starvation was stimulated by the aftermath of World War II. It

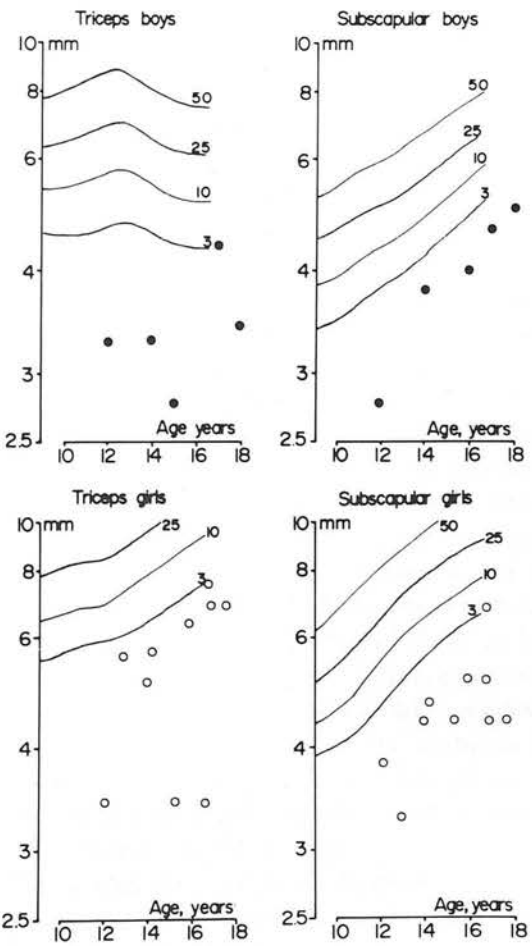

Fig. 2. Triceps and subscapular skinfolds in anorexia nervosa patients. 50th, 25th, 10th and 3rd percentile from Tanner et al. (32).

Table 2. *Regression equations for whole body potassium (^{40}K) on body weight, height and lean body mass (LBM) calculated from skinfold thickness in anorexia nervosa patients*

b is the regression coefficients of *x*, *a* is the constant term, *r* the correlation coefficient and *n* the number of patients

	n	*Y*	*b*	*x*	*a*	*r*	*p*
Boys	5	K	1.23	Weight	33.3	0.97	<0.01
Girls	10	K	1.41	Weight	24.6	0.80	<0.01
Boys	5	K	0.76	Height	−45.3	0.98	<0.01
Girls	10	K	0.63	Height	−27.1	0.45	>0.05
Boys	5	K	1.48	LBM	27.2	0.97	<0.01
Girls	10	K	1.84	LBM	15.0	0.75	<0.05

culminated in the classic Minnesota experiment from 1950 (26) which still remains today the standard reference for the biology of human starvation, though the study was confined to young adult men. AN is a severe form of starvation which differs in several respects from involuntary starvation (11). AN patients do eat small amount of food selectively, there are no signs of protein malnutriton, haemoglobin levels are usually normal and symptoms of vitamin deficiency diseases are rare. The degree of starvation or weight loss in our series are comparable or greater than those found in either famine victims or the laboratory experiments on semistarvation (26). The decrease of body weight in AN is not solely due to the loss of fat as our measurements of ^{40}K and estimates of FFW and LBM indicate (Table 1). In absolute terms, ^{40}K, LBM and BV are markedly reduced in the patients compared with normal children of the same age, but it should be noted that the reduction of BV and ^{40}K is in relation to their loss of body weight. The mean figure of 78 ml BV per kg of body weight compares favourably with normal data for healthy children (24). A similar proportion of diminuition of exchangeable potassium has been reported in adult patients (27) and in malnourished children (29). K is linearly associated with weight (Table 2). Similar relationship can be obtained from our data for BV ($r=0.60$), $p<0.05$). Further it would seem (Fig. 3) that the degree of emaciation in terms of loss of LBM or FFW can be estimated both from simple skinfold measurments and from determinations of ^{40}K. This is contrary to the view recently reported by Edmonds et al. (20). They found in 12 malnourished adult patients, 7 of whom had established AN that FFW determined from ^{40}K was low and the calculated fat values were high (range 20–50% body weight) and bordering on those found for the obese. It is difficult to reconcile their observations with our own results. It is well known that age, sex and physical fitness (8, 9) have an effect on ^{40}K and it is to be expected that ^{40}K will be reduced in AN as we and Edmonds et al. have found due to loss of cellular mass. Our ^{40}K values expressed in g per kg body weight are 2.12 for the boys and 2.10 for the girls, and compare favourably well with those of others (1, 7, 19). The validity of the determination of FFW from ^{40}K of course depends on the calculations used to obtain potassium content of human lean tissue. Edmonds et al. used aver-

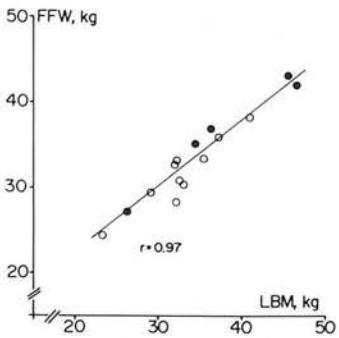

Fig. 3. Fat free weight (FFW) estimated from measurements of ^{40}K in relation to lean body mass (LBM) estimated from skinfold thickness. Symbols as Fig. 1. FFW=6.08+0.80 LBM: $p<0.001$.

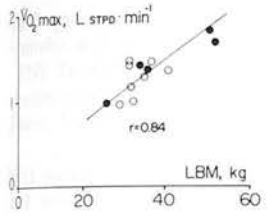

Fig. 4. Maximal oxygen uptake ($\dot{V}_{O_2}$ max) in relation to LBM estimated from skinfold thickness. Symbols as Fig. 1. $\dot{V}_{O_2}$ max=0.035+0.036 LBM; $p<0.001$.

age values and are open to some criticisms as are our own estimates which were made on a young but homogenous population of normal men and women and includes estimates of skeletal weight. The use of either their or our constants for the FFW equivalent of ^{40}K does not materially alter the two sets of data and the difference between the absolute values of ^{40}K in Edmonds et al.'s anorexic female patients and our own remain obscure. The calculation of LBM could also be critisized since it was derived from a normal material of 8 to 12-year-old children. However, there were no statistically significant difference between LBM calculated from the sum of two or ten skinfold thicknesses derived from children of similar age (30). The prediction of LBM from skinfold measurements in this study is in good agreement with other studies in young children (28) and in adults (5).

The loss of body weight and LBM is accompanied by a reduction in physiological work performance in AN. In the male and female patients absolute $\dot{V}_{O_2}$ max is decreased by approximately 45 and 40% respectively when compared to normal children of the same age (33). If account is taken of body weight, the difference between normals and patients is reduced to 30% for the boys and 20% for the girls. In relation to LBM the reduction, however, still seems to be of the order of 40% in the boys and 30% in the girls (30) with a mean value of 37.6 ml and 37.8 ml/kg LBM in boys and girls respectively. The decrease of $\dot{V}_{O_2}$ max is therefore greater than can be attributed to their change in body size and composition. However, some caution is necessary when interpreting data for $\dot{V}_{O_2}$ max. In the present study it was not always possible to apply the accepted criterion for $\dot{V}_{O_2}$ max namely a plateau of $\dot{V}_{O_2}$ on work load at exhaustion. However, the patients were encouraged to pedal the ergometer at maximal effort and the high levels of lactic acid concentration in their blood (21) bear witness to the fact that they did exercise to exhaustion and were at or very close to their "true" $\dot{V}_{O_2}$ max. The decrease in aerobic power is also in good agreement with results obtained from experimantal starvation (26). Undoubtedly as the disease progresses, and particularly during its later stages AN has a debilitating effect on the patients which must contribute to their low aerobic capacity. This in turn reduces their exercise tolerance still further and possibly give rise to a situation as found in the present investigation where $\dot{V}_{O_2}$ max decreases out of proportion to changes in body composition and as shown by Fohlin et al. (21) to the dimensions of the O_2 transporting system.

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Effects of Atropine and β-Blockade on Temperature Regulation and Performance during Prolonged Exercise

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Summary. The effects of intravenous injections of Atropine (1.8 mg) and practolol (15 mg) on the thermoregulatory responses to 1 h of exercise on a motor-driven treadmill have been investigated on six healthy subjects.

The results show that β-blockade had little effect on thermal responses to work except for a small but significant ($p < 0.05$) decrease in mean skin temperature ($\bar{T}_{sk}$) and peripheral tissue heat conductance (K). Metabolic (M) and total heat (H) production, and evaporative sweat loss (E) and rectal temperature (T_{re}) were similar to control values. In contrast, atropine, particularly at work loads beyond 60% maximal aerobic power output ($\dot{V}O_{2\ max}$), raised T_{re} ($p < 0.001$), $\bar{T}_{sk}$ ($p < 0.001$) and reduced E by approximately 50%. At the highest work loads T_{re} increased as a linear function of time during the latter part of exercise, and at the 60th min was almost independent of relative stress (expressed as % $\dot{V}O_{2\ max}$) imposed on the subjects. At the lower work loads the majority of subjects reached thermal equilibrium before the end of exercise by maintaining their convective heat transfer from core to periphery by increasing peripheral blood flow (as indicated by K), and raising their heat losses to environment by convection and radiation. The latter pathways for heat dissipation were enhanced by the subjects ability to sustain a $\bar{T}_{sk} \sim 4°$ C above control values independently of M. Atropine had no effect on M or H but greatly affected work performance, no subject was able to exercise at loads > 70% $\dot{V}O_{2\ max}$ for 1 h. These results demonstrate the ability of the thermoregulatory system to adapt to β-adrenergic and to parasympathetic blockade during light exercise, and underline the effects of a reduction in the capacity of the sweating mechanism on physiological performance at higher rates of work.

Key words: Atropine – Practolol – Rectal temperature – Sweat rate – Physiological performance.

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List of Abbreviations used in the Text

M = Metabolic heat production
H = Total heat production
E = Evaporative sweat loss
T_{re} = Rectal temperature
$\bar{T}_{sk}$ = Mean skin temperature
K = Peripheral tissue heat conductance
PBF = Peripheral blood flow
$\dot{V}O_{2\,max}$ = Maximal aerobic power output
f_H = Cardiac frequency

Several authors have investigated the effects of parasympathetic (PSB) and β-blockade on cardiovascular haemodynamics during short-term dynamic exercise in normal subjects. Ekblom et al. [5] found that atropine raised cardiac frequency above control conditions, its effect being diminished at progressively higher work loads. A compensatory reduction in stroke volume, maintained cardiac output within normal limits. β-Blockade induced by propranolol [6, 7] and practolol [10, 12] reduces cardiac frequency and output while maintaining the same or slightly higher stroke volume and arteriovenous oxygen content difference.

In both types of blockade the oxygen cost of work, and the maximum working capacity were unchanged or marginally lowered [5]. However, no studies have examined the effects of atropine, practolol or propranolol on the physiological response to prolonged exercise. This is of particular importance when one considers that in addition to the haemodynamic effects outlined for the three drugs, atropine affects the dissipation of heat by partially blocking the release of sweat from apocrine glands.

In the present study we have examined the separate effects of atropine and practolol on oxygen transport and thermoregulation during prolonged exercise in six healthy men.

Table 1. Physical characteristics of the six healthy male subjects who took part in the investigation. Age, weight, height, and maximal aerobic power output ($\dot{V}O_{2\,max}$)

Subject	Age (year)	Weight (kg)	Height (cm)	($\dot{V}O_{2\,max}$) (l/min)
1	22	64.3	175.2	3.20
2	21	71.5	178.7	3.30
3	42	62.0	178.0	3.50
4	35	63.9	174.0	4.80
5	26	72.5	173.0	4.10
6	21	70.0	189.4	4.16

Material and Methods

The physical characteristics of the subjects are given in Table 1. Each subject was required to exercise at several different levels of exercise for 1 h on a motor-driven treadmill. They were weighed nude and clothed in shorts and plimsolls before exercise, and had an indwelling needle inserted into their antecubital vein. At least 10 min before exercise they were injected with either 1.8 mg atropine or 15 mg practolol. During the hour of exercise frequent measurements of oxygen intake ($\dot{V}O_2$) and skin temperature at 13 sites were made, and rectal temperature (T_{re}) and cardiac frequency (f_H) were measured continuously as described elsewhere (Davies et al. [3]). At the end of exercise they were reweighed. Standard calculations of metabolic (M), and total heat (H) production, mean skin temperature ($\bar{T}_{sk}$); evaporative sweat loss (E) and peripheral tissue heat conductance (K) were made from the raw data. This applied to all experiments performed except for those with atropine at work loads greater than 60% maximal aerobic power output ($\dot{V}O_{2\ max}$). Under these conditions the subjects were not able to achieve thermal equilibrium during the final 15 min of exercise, thus precluding calculation of K. Maximal aerobic power output ($\dot{V}O_{2\ max}$) was measured on a separate occasion for control conditions using the criteria and method described by Davies [2].

Results

A comparison of the effects of β-blockade and atropine with the normal thermal and metabolic responses to exercise is given in Tables 2 and 3.

It can be seen that practolol has little effect on the thermoregulatory responses to exercise (Table 2) except that it produces a slight (but significant, $p < 0.05$) reduction in $\bar{T}_{sk}$ and K. For a given H and M respectively, E and T_{re} lie within the normal range of control values. Similarly (Table 3) atropine is without effect on M or H or T_{re} at low levels of work, but in contrast to the administration of practolol, it significantly raises $\bar{T}_{sk}$ ($p < 0.001$) and K ($p < 0.001$) and reduces E. At high work levels (> 60% $\dot{V}O_{2\ max}$) these significant differences persist (except for K, which cannot be calculated under these conditions – see methods); and atropine effects a marked rise in T_{re} for a given M. Indeed, between 65 and 70% $\dot{V}O_{2\ max}$ (Fig. 1) T_{re} increases during the latter part of work as a linear function of duration, and at the 60. min is

Table 2. The effects of β-blockade on metabolic thermoregulatory function. Metabolic (M) and total (H) heat production; rectal (T_{re}) and mean skin ($\bar{T}_{sk}$) temperature; evaporative sweat loss (E) and peripheral tissue heat conductance (K)

		% $\dot{V}O_{2\ max}$	M Watts	H Watts	T_{re} °C	$\bar{T}_{sk}$ °C	E Watts	K Watts °C^{-1}m^{-2}
Control	Mean	62.0	812	691	38.76	29.97	388	34.4
($n = 6$)	S.D.	± 9.0	± 220	± 191	± 0.35	± 0.68	± 237	± 12.1
β-blockade	Mean	62.5	815	698	38.63	30.98[b]	394	41.3[a]
($n = 6$)	S.D.	± 6.4	± 191	± 172	± 0.25	± 0.73	± 149	± 9.8

Significance: [a] $p < 0.05$; [b] $p < 0.01$

Table 3. The effects of atropine on thermoregulatory function (a) below 60% $\dot{V}O_{2\,max}$ and (b) above 60% $\dot{V}O_{2\,max}$ (abbreviations see Table 2)

		% $\dot{V}O_{2\,max}$	M Watts	H Watts	T_{re} °C	$\bar{T}_{sk}$ °C	E Watts	K Watts °C^{-1}m^{-2}
(a) below 60% $\dot{V}O_{2\,max}$								
Control	Mean	56.2	739	638	38.38	30.86	326	36.5
($n = 5$)	S.D.	± 3.0	± 42	± 27	± 0.26	± 0.29	± 124	± 4.1
Atropine	Mean	55.7	732	624	38.54	34.78[b]	183	78.3[b]
($n = 5$)	S.D.	± 2.6	± 49	± 26	± 0.38	± 0.85	± 31	± 17.2
(b) above 60% $\dot{V}O_{2\,max}$								
Control	Mean	66.8	822	698	38.71	31.53	388	41.9
($n = 5$)	S.D.	± 5.7	± 98	± 76	± 0.34	± 0.80	± 163	± 7.9
Atropine	Mean	66.1	813	691	39.37[a]	34.85[c]	200[b]	–
($n = 5$)	S.D.	± 5.3	± 106	± 85	± 0.82	± 0.52	± 56	–

Significance: [a] $p < 0.05$; [b] $p < 0.01$; [c] $p < 0.001$

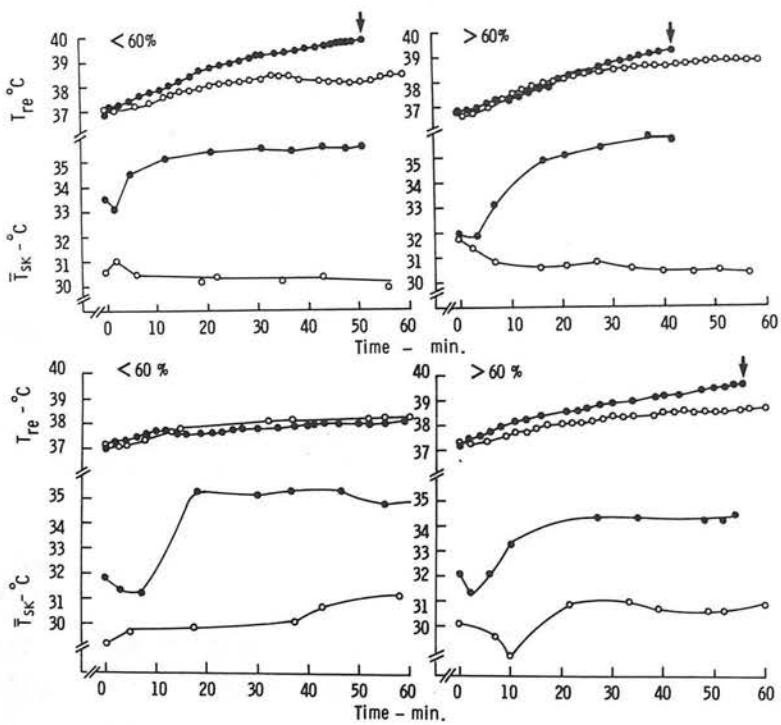

Fig. 1. Time course of rectal (T_{re}) and mean skin ($\bar{T}_{sk}$) temperature changes during prolonged exercise. **(a)** An athletic Subject (4) who was unable to complete 1 h of exercise at work loads > 60% $\dot{V}O_{2\,max}$ and **(b)** Subject 5. (●) Atropine; (○) control. The arrow indicates the time at which the subject became exhausted and could not continue the exercise

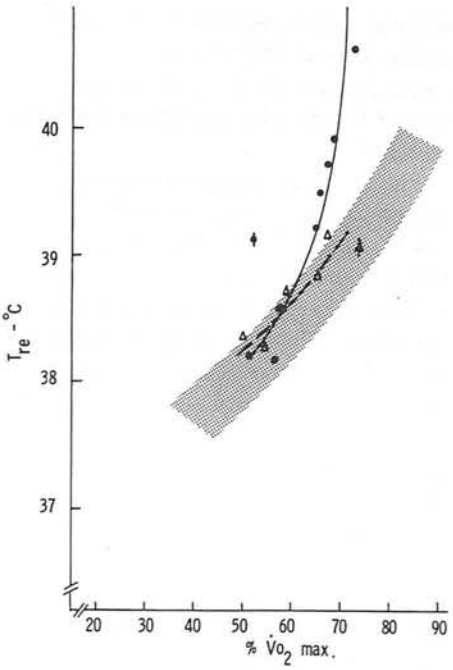

Fig. 2. The relationship between rectal temperature (T_{re}) and relative aerobic power output (% $\dot{V}O_{2\ max}$). (●) Atropine; (△) β-blockade. Shaded area represents limits (mean ± SD) found previously (Davies et al. [3]) for subjects exercising under normal conditions. Subject 4 is indicated separately with a vertical line across the appropriate symbol

almost independent of relative work load (Fig. 2). The effect of atropine on the T_{re} of the athletic subject [4] was more marked and occurred at a lower % $\dot{V}O_{2\ max}$ (Fig. 2), his $T_{re}/\dot{V}O_{2\ max}$ points being displaced to the left of those found for the more sedentary subjects.

Discussion

Ekblom et al. [5] have reported that β-blockade and atropine are without effect on aerobic metabolism during short term exercise. We are able to confirm this finding for practolol but in contrast to the data of Ekblom et al. we found that maximal performance during prolonged work was greatly restricted with atropine (Tables 2 and 3).

Administration of practolol reduced $\bar{T}_{sk}$ ($p < 0.5$), but E for a given H, and T_{re} for a given % $\dot{V}O_{2\ max}$ (Fig. 2) were within the normal range. All subjects were able to exercise over their complete range of work capacity without undue stress or ill effect, and said they felt cooler and more comfortable during exercise with β-blockade. They worked with a heart rate of 20 beats · min^{-1} lower than in the control experiments. The thermoregulatory effects of practolol are similar to those previously found for hypoxia [8] but without the deleterious effect on $\dot{V}O_{2\ max}$. Since β-blockade reduced $\bar{T}_{sk}$, the $T_{re} - \bar{T}_{sk}$ gradient at a given H increased, and thus K was reduced. This would indicate that the small reduction in $\dot{Q}$ found with practolol administration [10] has little effect on thermoregulatory function except a slight

fall in K which suggests a reduced blood flow to the skin [11]. However, only small doses of practolol were administered in these experiments and we do not know if larger doses of the drug would have produced a more dramatic effect on thermoregulatory function by effecting a more complete β-blockade (Sutton, personal communication).

In contrast to practolol, atropine had a marked effect on physiological performance during prolonged work. It reduced sweating significantly ($p < 0.001$) by $\sim 47\%$, raised $\bar{T}_{sk}$ and T_{re}, and produced muscular weakness and signs of hyperthermia at work levels beyond 60% $\dot{V}O_{2\ max.}$ No subject was able to exercise beyond $\sim 70\%$ of his work capacity for 1 h, and the effects of the drug were more severe in the athlete (Figs. 1 and 2). At low work loads the sedentary subjects (and particularly those who were thin) seemed able to compensate for their decreased sweat loss by increased convection and radiation from the skin. The high $\bar{T}_{sk}$ which was maintained independently of M (Table 3) allowed the subjects to increase the heat loss by radiation and convection. They completed the work with a normal rectal temperature (Fig. 1) and without discomfort. At high work loads this strategy was ineffectual. Above $\sim 60\%$ $\dot{V}O_{2\ max}$, the T_{re} of all subjects rose as a linear function of time (Fig. 1), and the exercise was usually stopped voluntarily, or by the observer if heart rate, (dis) comfort rate and the rated perceived exertion were at a maxima. Clearly with sweating reduced by almost half, collapse from anhydrotic heat exhaustion [1] was to be expected, but interestingly no subject complained or suffered from heat pyrexia per se even though rectal temperatures in excess of 40° C were recorded in two subjects at the end of exercise (Fig. 2). The major complaints were of muscular tiredness and weakness, an extremely hot skin, and peripheral turgor, particularly in the fingers, which were sometimes painful. It would seem therefore that at low work loads the body can maintain thermal equilibrium with the environment despite the sweat suppression; at high levels the thermoregulatory system is overwhelmed, and body heat stores and T_{re} rise rapidly.

Since it is well established [13] that sedentary subjects exercise with a higher T_{re} but same $\bar{T}_{sk}$ for a given M than athletes we have argued in a previous paper [3] that the rise in T_{re} during exercise is related to M and the cardiovascular capacity of the individual. The latter will govern the distribution of $\dot{Q}$ and the convective transfer of heat from the core to periphery. To meet the requirements of the Fick equation applied to heat flow (see [11]) peripheral blood flow (PBF) must vary as a reciprocal function of the $T_{re} - \bar{T}_{sk}$ gradient.

These findings would appear to hold for the present experiments. In β-blockade with practolol $\bar{T}_{sk}$ is held at a lower level, and K is reduced (Table 2) indicating a lower PBF due possibly to a reduction in cardiac output [4, 8]. Atropinisation has the opposite thermal effect, but again $\bar{T}_{sk}$ is held constant (but at a higher level) and independently of M. The inhibition of sweating at work loads $< 60\%$ $\dot{V}O_{2\ max}$ reduces the $T_{re} - \bar{T}_{sk}$ gradient and increases K, and therefore the decreased evaporative heat loss is compensated for by an increased PBF and a rise in $C + R$. At higher work loads this strategy is still maintained, but since $\bar{T}_{sk}$ is held constant and seemingly not allowed to rise beyond $\sim 35.5°$ C (Fig. 1 and Table 3), the available heat dissipating mechanisms become saturated. The subject becomes effectively "poikilothermic" and T_{re} rises as a linear function of the duration of exercise. Thus in general the thermoregulatory system would appear to operate from a baseline of constant

$\bar{T}_{sk}$ for a given condition and maintains this by precisely coordinating the convective heat transfer from the core to the periphery with the evaporative and $C + R$ heat losses from the skin to the environment. The core temperature is then seen as a function of M and PBF. If the evaporative sweat loss to the environment is suppressed as in the present experiments, $\bar{T}_{sk}$ will rise and the $T_{re} - \bar{T}_{sk}$ gradient will be reduced, necessitating a rise in PBF if the requirements of heat convection from core to periphery are to be met. Clearly this mechanism must ultimately be self-restricting since the displacement of blood volume to the periphery must be compatible with the maintenance of systemic blood pressure and muscle blood flow (if exercise is to continue). At low work levels a rise in $\bar{T}_{sk}$ will result in increased $C + R$ losses which may compensate for a decreased E, but at higher work intensities $C + R$ will remain constant (since T_{sk} is independent of M — Table 3), PBF will reach a maximum, and further 'active' control of thermoregulation will be impossible. The body heat stores will increase and result in an uncompensated rise in T_{re} (Fig. 1).

Thus the present experiments underline the importance of the coordination of the convective heat transfer to the skin, with the evaporative (and $C + R$) losses from it, but they in no way identify the underlying controlling mechanism(s) during work. Recently Rowell [13] has suggested that both *E and PBF* are under common (core temperature) control but it is difficult to reconcile their findings with the present data. Atropine and practolol had opposite effects on both $\bar{T}_{sk}$ and PBF (as indicated by K) at work loads below 60% and yet for given M, T_{re} was the same. Under these circumstances one must either suggest that the sensitivity of the PBF control mechanism is changed by the drugs or factors other than central temperature are responsible (as found in animal experimentation [9]), for the regulation of the two main effector heat dissipating channels during exercise in man.

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An Assessment of a N_2O Rebreathing Method for the Estimation of Cardiac Output During Severe Exercise

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Cardiac output was measured as pulmonary blood flow by a non-invasive N_2O rebreathing technique during exercise at submaximal and maximal work levels in three subjects. The standard deviation of repeated measurements over a period of 6–20 weeks was $\pm 1{\cdot}5$ l min^{-1} at all levels of work and in all subjects. This level of variability is comparable to that found in using the dye dilution and direct (O_2) Fick methods in individual subjects during a more limited time span, and suggests that the method can be used effectively in non-steady state and maximal conditions of dynamic exercise.

1. Introduction

The direct Fick and dye dilution methods are often used to measure cardiac output during exercise but they require stable or 'steady state' physiological conditions as a criterion for their use. The direct Fick equation would require modification in order to apply to non-steady state conditions. For this reason we have investigated the accuracy and reproducibility of a non-invasive 'foreign gas' (nitrous oxide) rebreathing method for the measurement of cardiac output at high work intensities. The N_2O method estimates cardiac output (as pulmonary blood flow) by using a form of the Fick equation developed by Bornstein (1910) in which all the necessary parameters are obtained within the rebreathing period.

2. Material and Methods

Three moderately fit male adults (26–41 yr old) acted as subjects for the study and were fairly similar in height (169–178 cm) and weight (62–68 kg). All studies were performed on a bicycle ergometer placed in the sitting position, and experiments were carried out over a period of six weeks (Subjects RR, CTMD) to 20 weeks (Subject EZ). Two-leg pedalling exercise was performed on an *eletrically braked bicycle* (*Elema-Schonander*) by all subjects, while a specially modified *friction belt bicycle* (*Monark*) was used for arm, and combined arm and leg cranking exercise in subject EZ.

In each subject the maximum working capacity was determined as the maximum work load sustained for a period of four minutes, and on subsequent occasions subjects performed progressively harder work loads up to and above the maximum working capacity. A period of 3·5 minutes exercise was required at the highest work load, and oxygen consumption was determined at the last 50 s of the test, followed by a 10 s nitrous oxide rebreathing manoeuvre to determine cardiac output. The duration of exercise at lower work loads was related to the severity of stress, but generally, gas exchange and cardiac output measurements were made at 6–7 min intervals for periods not exceeding 30 min. During an experimental day repeated exercise tests were combined with rest periods of 0·5–1 hour.

In all tests the subjects breathed through a *low resistance 2-way valve*, and at intervals a *Douglas bag* was used to collect expired gas for gas exchange measurements. Expired volume was measured on a *gas meter* (*Parkinson Cowan CD4*) while fractional

cranking did not differ significantly from normal 2-leg cycling. Pulmonary recirculation time was reduced and approached an asymptotic value of ~7·5 s at high work loads (Figure 2). The rise in cardiac output at high work loads was exclusively achieved by an increase in cardiac frequency.

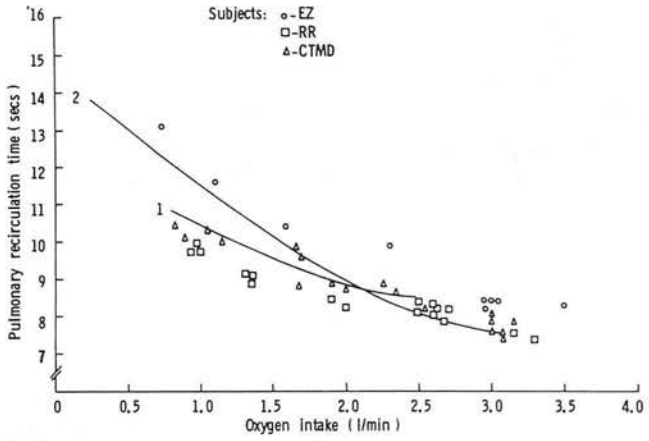

Figure 2. Recirculation time at various work loads, plotted against oxygen intake (STPD) for subjects EZ (○), RR (□) and CTMD (△). Regression lines 1: for the data of Rigatto *et al.* (1968) and 2: ZeidiFard *et al.* (1976) are also included.

4. Discussion

The present study confirms and extends our previous findings obtained at submaximal levels of work (ZeidiFard, Godfrey and Davies 1976). The investigation shows that cardiac output can be estimated with an accuracy of $\pm 1{\cdot}5\,l\,min^{-1}$ independently of work load up to and including maximal exercise (Figure 1). Nevertheless the estimation of cardiac output using the N_2O rebreathing technique during severe work may be queried in several respects. For example, at high work loads estimates may be low due to the occurrence of recirculation before the 8–10 s period required for the N_2O rebreathing manoeuvre. However, in all subjects recirculation time was reduced asymptotically with oxygen intake, and was not less than 7·5 s even at the highest work loads (Figure 2). These observations are consistent with our earlier findings (ZeidiFard, Godfrey and Davies 1976) and the present estimates of recirculation time are comparable to the values obtained by the dye dilution techniques (Sowton *et al.* 1968).

Some workers (*e.g.* Chapman *et al.* 1950 and Donevan *et al.* 1962) have argued that hyperventilation may affect rebreathing estimates of cardiac output by raising $\dot{V}_{O_2}$ or $\dot{Q}$ or both variables simultaneously. The increases in $\dot{Q}$ due to hyperventilation found by Donevan *et al.* 1962 were small and well within the error of the dye dilution method of measuring cardiac output which they used. Furthermore since our subjects performed severe exercise the error due to hyperventilation would tend to diminish rather than increase. The correction applied by Chapman *et al.* 1950 for the difference between rebreathe and steady state values of $\dot{V}_{O_2}$ measured at rest has been investigated by Rigatto, Jones and Campbell 1968. They found that at work loads in excess of 300 kpm min^{-1} cardiac frequency and oxygen consumption measured during the rebreathe period were similar to the values noted prior to it, so that a correction of the measured cardiac output was unnecessary. Pugh (1972) came to a similar conclusion

for subjects doing severe exercise, but based his argument on the small changes in the O_2/CO_2 content of blood which were induced by alteration in the fractional gas concentrations of O_2 and CO_2 during the rebreathing manoeuvre. Observations in the present study confirm the view held by these authors.

In relation to oxygen intake cardiac output increased curvilinearly (Figure 1), beyond $\sim 40\%$ $\dot{V}_{O_2\,max}$. Stroke volume did not increase significantly. Similar relationships have been reported by others (Åstrand *et al.* 1964), though it should be noted in our results that the degree of non-linearity is subject dependent and not necessarily statistically significant.

Work loads in excess of the maximal oxygen intake during leg-pedalling and arm-cranking (where the maxima in oxygen intake and cardiac output occurred at much lower work loads) did not elicit further increases in cardiac output. The maximal oxygen intake and cardiac output during leg-pedalling could not be increased by using combined arm and leg exercise (see Stenberg *et al.* 1967), so that although the results suggested that the type of work limited $\dot{V}_{O_2\,max}$ and cardiac output, there was no evidence to suggest that $\dot{V}_{O_2\,max}$ was limited by the cardiac output. Ouellet, Poh and Becklake (1969) have claimed that the latter is the case during maximal leg exercise on a bicycle ergometer, however, the reproducibility of their steady state N_2O inhalation method would make such a conclusion contentious. Furthermore, Hermansen, Ekblom and Saltin (1970) have shown that both cardiac output and $\dot{V}_{O_2\,max}$ could be raised a further 6% by changing the type of exercise from cycling to treadmill running.

5. Conclusions

The N_2O rebreathing method can be used to measure cardiac output, as pulmonary blood flow, with a reproducibility comparable to that of the dye dilution and direct (O_2) Fick methods. In contrast to these methods the N_2O rebreathing procedure is non-invasive so that its use over an extended period of time and in various work situations in individual subjects need not be restricted. Unlike the acetylene method, the N_2O method allows continuous analysis of rebreathed gases, so that the adequacy of mixing and subsequent recirculation can be measured rather than assumed. Estimates of cardiac output were found to be equally reproducible during submaximal steady state exercise, and during high to maximal unsteady state exercise on a bicycle ergometer; the method is therefore potentially useful in other forms of unsteady state exercise. Measurements of cardiac output during maximal exercise with different muscle groups indicated that the type of exercise itself determined both cardiac output and oxygen intake.

We wish to thank Miss G. Preston, Mr. E. E. Davies and Mr. D. Rogers for technical assistance, and Mr. R. Roberts for acting as a subject. We are grateful to the Department of Respiratory Medicine, Hammersmith Hospital for providing their laboratory facilities.

Le rendement cardiaque a été déterminé à partir du flux sanguin pulmonaire par une technique de re-inhalation du N_2O non-envahissante, durant un exercice au niveau de travail maximal et submaximal effectué par trois sujets. L'écart-type obtenu au cours d'évaluations répétées sur une période de 6 à 20 semaines était de $\pm 1{,}51\ mm^{-1}$ pour tous les niveaux de travail et pour tous les sujets. Ce degré de variabilité est comparable à celui utilisant les méthodes de la dilution et directe (O_2) de Fick sur des sujets individuellement pendant une période plus courte et suggère que la méthode peut être utilisée pour un régime non constant et des conditions maximales d'un exercice dynamique.

Bei drei Versuchspersonen wurde die Herzleistung aus der pulmonaren Durchblutung unter Verwendung einer nichtangreifenden N_2O Beatmungstechnik bei maximaler und submaximaler Arbeit ermittelt. Die Standardabweichung von wiederholten Messungen während einer Periode von 6 bis 20 Wochen betrug $\pm 1{,}5$ Liter pro Minute für alle Arbeitsschweren und alle Versuchspersonen. Diese Variabilität kann mit Ergebnissen von Untersuchungen verglichen werden, bei denen die direkte (O_2) Methode von Fick an einzelnen Versuchspersonen innerhalb einer begrenzteren Untersuchungszeit angewendet wurde. Es wird nachgewiesen, daß die Methode wirkungsvoll in nicht-steady state Untersuchungen und bei maximaler dynamischer Arbeitsbelastung angewendet werden kann.

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The Effects of Different Levels of Heat Production Induced by Diathermy and Eccentric Work on Thermoregulation During Exercise at a Given Skin Temperature

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Summary. The thermal responses of two healthy male subjects have been studied at the same mean skin temperature ($\bar{T}_{sk}$) during negative work, positive work and positive work in which additional heating was induced by diathermy. The results showed that for a given metabolic heat production (M) rectal (T_{re}) and oesophageal (T_{oes}) temperatures were higher in negative work and positive work with diathermy than normal control experiments. In resting experiments with diathermy, T_{oes} rose to the same level as when an equal amount of heat was produced metabolically by exercise. In negative work and positive work with diathermy sweat loss ($\dot{M}_{sw}$) was higher for a given M and $\bar{T}_{sk}$ than found for normal exercise, but in all three forms of work the relationship of $\dot{M}_{sw}$ to total heat production (H) was identical. During positive work with and without diathermy the differences in $\dot{M}_{sw}$ could be accounted for by using a previously developed model of relative sweating rate: $\%\dot{M}_{sw} = -$ constant $+ \alpha T_{re}$ (or T_{oes}) $+ \beta \bar{T}_{sk}$.

In negative work, removal of the difference between predicted and observed $\%\dot{M}_{sw}$ required the inclusion of a further factor into the equation based on muscle temperature. The results suggest that the core temperature in exercise rises to meet the requirements of heat dissipation mainly by stimulating $\dot{M}_{sw}$ and establishing a heat transfer gradient from core to periphery and is not necessarily or uniquely related to M or to the rate of working. The study underlines the usefulness of negative work and diathermy as physiological tools for the further understanding of thermoregulation during exercise.

Key words: Temperature – Exercise – Rectal temperature – Skin temperature – Sweat loss – Short wave radiation – Negative work

It is now well recognised that rectal temperature (T_{re}) rises during exercise to a level which is independent of environmental conditions over a wide range (Lind, 1963). Nielsen (1938) who was the first person to describe this phenomenon suggested that

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the mechanism regulating T_{re} must be set by the work output. Since in Nielsen's study the rate of work was closely related to the heat production and the latter in the steady state of exercise was equal to the heat loss, Kerslake (1972) has suggested an equally plausible interpretation, namely, that "the rate of heat loss is controlled at a level which is dependent on the core temperature". Both Nielsen's and Kerslake's theories are valid provided the rate of working and rate of heat loss are closely linked which is usually the case under most conditions of exercise. However, the interdependence between the two variables can be broken by the use of negative (eccentric) work during which energy is absorbed by the contracting muscle and later dissipated as 'extra' heat. This form of work has been studied several times but unfortunately no definitive conclusions can be drawn from the available data regarding the opposing theories of Kerslake and Nielsen. For example, Nielsen (1966) found oesophageal temperature (T_{oes}) to be the same in negative and positive work whereas Smiles and Robinson (1971) reported lower T_{re} and Nadel et al. (1972) higher T_{oes} values when their subjects performed eccentric work at given metabolic rate (M). Further, Nadel and his co-workers found mean skin temperature ($\bar{T}_{sk}$) to be 3° C higher in negative than positive work at the same ambient temperature whereas Nielsen et al. (1972) found $\bar{T}_{sk}$ to increase linearly with environmental temperature and to be independent of the type of work performed, though they did find that the upper limit for thermal equilibrium was reduced in negative work. The common observation in all four studies was that sweat loss ($\dot{M}_{sw}$) was closely correlated to total heat production (H) independently of the way exercise was performed. Nielsen (1966) concludes that thermoregulation in negative work can only be understood if we accept that the core temperature is set at a level proportional to M and not to the requirements of heat dissipation. On the other hand Nadel et al. (1972) feel that thermoregulatory control during exercise presents no problems and the same close association of $\dot{M}_{sw}$ to H in both negative and positive work can be resolved if account is taken of the different levels of thermal inputs. In other words, whereas Nielsen's interpretation of her data denies the Kerslake theory, the work and view of Nadel and his co-workers are entirely consistent with a model of thermoregulation related to requirements of heat dissipation. However, it should be stated that Smiles and Robinson (1972) found a low correlation between $\dot{M}_{sw}$, $\bar{T}_{sk}$, and T_{re} and suggest stimuli of neuromuscular origin may be implicated in thermal control during exercise which varies in type and intensity.

The second method which may be used to dissociate the rate of working from the rates of heat production and heat loss is the use of short wave diathermy. Again Nielsen and Nielsen (1965) have investigated this technique by performing experiments in which a given amount of heat was diathermically induced into the body at rest and then compared to the thermal responses in the same subjects producing a similar amount of heat metabolically during exercise. They found that T_{re} rose to the same level independently of whether the heat was metabolically produced or received from the diathermy apparatus. At a given $\bar{T}_{sk}$, $\dot{M}_{sw}$ was the same in both procedures which suggests that the rise in T_{re} is not uniquely associated to the rate of working but is related to requirements of heat dissipation. The results they obtained were essentially the opposite to Nielsen's observations the following year in negative work, but this did not appear to influence the interpretation of her later findings.

In order to try and resolve these differences in results found during negative work and the apparently contradictory observations of Nielsen, the effects of induced heat by diathermy at rest and during exercise have been investigated and the results compared in the same subjects producing the same and greater amounts of heat during negative and positive work at the same $\bar{T}_{sk}$ on the motor-driven treadmill.

Material and Methods

Two healthy subjects were used in the experiments (CTMD, age 44 years, weight 62.0 kg, height 178 cm, A_D 1.78 m^2 and CSB, age 22 year, weight 66.5 kg, height 180 cm, A_D 1.85 m^2), who were almost identical with regard to their maximal aerobic power outputs. CSB performed the negative work experiments at two different ambient temperatures, T_{db} 21 and 15° C with an air flow 2 m/s and T_{wb} of 15 and 10° C, respectively. The experiments were performed in an air-conditioned room in which a given air temperature could be maintained for the period of the experiment to within ± 1° C. The positive work experiments were designed to match the mean skin temperature ($\bar{T}_{sk}$) found in the negative exercise. This was achieved by raising, if necessary (see Table 1), the room and $\bar{T}_{sk}$ temperature by radiant heat. The diathermy experiments were performed by CTMD. The short wave heating was induced through pads placed on the anterior and posterior sides of the thighs of each leg. The pads were connected by induction cables to an ultratherm apparatus (Siemens Ltd.). Observations were made at rest with the subject seated and at various levels of positive work on a motor driven treadmill. For this purpose the diathermy apparatus was housed on a specially constructed platform at the rear of the treadmill which enabled the subject to walk freely and so overcome the constraints imposed by the short (but necessary standard) cable length supplied with the apparatus. Care was taken to ensure that the cables did not touch each other or the subject by the use of plastic divider clips. The pads were housed in shorts which were tailor-made with pockets which adhered closely to the thighs. However, during exercise the movement of thighs in walking undoubtedly gave rise to changes in the force field compared to the resting condition in which the legs were immobile. This factor probably accounts in part for the lower amounts of heat that could be induced during work as compared to the rest (Table 2 and below).

The subjects reported to the laboratory each morning after a light breakfast and were fitted with the necessary electrodes (for recording ECG) and rectal probes and weighed nude and clothed before the commencement of exercise. During positive and negative work rectal temperature (T_{re}) was recorded continuously from the probe which was inserted 8 cm above the anal sphincter. Skin temperatures were recorded at the 15th, 45th, and 60th min of exercise and subsequently calculated (and weighed from 13 sites) to give a value of mean skin temperature ($\bar{T}_{sk}$) as previously described (Davies et al., 1976). Metabolic heat production (M) was calculated from measurements of oxygen intake measured by an open circuit technique between the 20th and 25th min and 50th and 55th min of exercise. Total heat production (H) was calculated from M and the vertical work (± W) performed based on the subjects body weight, speed of walking and inclination of the treadmill. Heat conductance (K) of the peripheral tissues was calculated after the method of Burton (1934). During the diathermy experiments core temperature was measured from an oesophageal (T_{oes}) probe to avoid the direct heating effects of the diathermy on the rectum and the thermister. Since the oesophageal site was away from the centre of the short wave field, it was found that the probe could be left in situ during exercise without ill effect. The recording apparatus was protected from the field at a distance by thick metal sheets. However, as a precaution the measurements of $\bar{T}_{sk}$ and T_{oes} were always taken with the diathermy momentarily switched off. Control experiments were performed in the same way except that both T_{re} and T_{oes} were recorded. The subject exercised with the pads attached to the thighs and the cables connected to diathermy apparatus which was left unplugged. Immediately on cessation of exercise in all three forms of work, the subjects were again weighed nude and clothed and from the weight changes during work sweat production ($\dot{M}_{sw}$) was calculated in the standard way with account being taken of respiratory and metabolic water changes. In the four diathermy and four separate negative work experiments, muscle temperature (T_m) was taken using a needle probe (Ellab. Ltd.) within 1 min of the end of exercise. The

total heat production (H) during the diathermy experiments (i.e., $M-W$ + the electrically induced heat) was calculated after the method of Nielsen and Nielsen (1965) from the heat balance equation: $H = C + R + E - \Delta S$, where C = convection heat loss, R = Radiation heat loss, E = evaporative heat loss, and S = change in body heat content: Heat exchange through convection (C) and radiation (R) was estimated according to the formulas derived by Kerslake (1972) C (w/m²) = $8.3\sqrt{V}(\bar{T}_{sk} - T_A)$ and R (w/m²) = $5.67 \times 10^{-8}(\bar{T}_{sk}^4 - T_r^4)$, where V = air velocity in m/s and T_r = radiant temperature in °K. The change in heat content was estimated from the equation: $S = 0.83\ W\ (0.9\ \Delta T_{re}$ and $0.1\ \Delta \bar{T}_{sk})$ where 0.83 is the specific heat of the body tissues in Kcal and W is body weight in kg and then converted to w/m². At rest the mean extra heat induced by diathermy was calculated to be 292 ± 17 W and during exercise 198 ± 25 W.

Results

The basic data for subjects CTMD and CSB in which $\bar{T}_{sk}$ was maintained at the same level in positive (+ *ve*) and negative (− *ve*) work and in positive work with and without diathermy are shown in Tables 1 and 2.

Table 1. Oxygen intake, metabolic (M) and total heat (H) productions, rectal (T_{re}) and mean skin ($\bar{T}_{sk}$) temperatures, sweat loss ($\dot{M}_{sw}$) and peripheral tissue heat conductance (K) during positive (+ W) and negative (− W) work at two different skin temperatures. Subject CSB. Mean (± SD) data for 16 experiments

Work	$\dot{V}O_2$ (l/min)	M (W)	H (W)	T_{re} (°C)	$T_{\dot{s}k}$ (°C)	$\dot{M}_{sw}$ w/m²	K w/m² °C
−W	1.35 ± 0.19	459 ± 66	834 ± 139	37.70 ± 0.13	28.70 ± 0.70	149 ± 40	42.9 ± 7.8
+W	1.33 ± 0.20	453 ± 67	432 ± 48	37.48 ± 0.18	29.31 ± 0.87	52 ± 17	27.8 ± 2.3
Significance	NS	NS	<0.001	<0.5	NS	<0.001	<0.01
−W	1.57 ± 0.27	534 ± 91	911 ± 165	38.05 ± 0.13	31.79 ± 0.39	243 ± 50	64.5 ± 11.2
+W	1.58 ± 0.30	530 ± 87	501 ± 69	37.66 ± 0.19	31.81 ± 0.18	138 ± 29	43.2 ± 5.5
Significance	NS	NS	<0.001	<0.001	NS	<0.001	<0.01

Table 2. Effects of diathermy at rest and during exercise. Mean (± SD) data for 14 experiments. (Symbols as for Table 1). Subject CTMD

	$\dot{V}O_2$ (l/min)	M (W)	H (W)	T_{oes} (°C)	$\bar{T}_{sk}$ (°C)	$\dot{M}_{sw}$ w/m²	K w/m² °C
Rest	0.29 ± 0.03	99 ± 11	391 ± 13	37.55 ± 0.06	30.28 ± 1.36	50 ± 9	30.7 ± 6.4
Control	1.88 ± 0.49	640 ± 170	574 ± 119	37.89 ± 0.46	29.19 ± 0.53	115 ± 46	30.6 ± 5.6
Diathermy	1.90 ± 0.49	647 ± 169	778 ± 106	38.20 ± 0.41	29.02 ± 0.68	213 ± 43	42.6 ± 1.5
Significance	NS	NS	<0.001	<0.01	NS	<0.001	NS

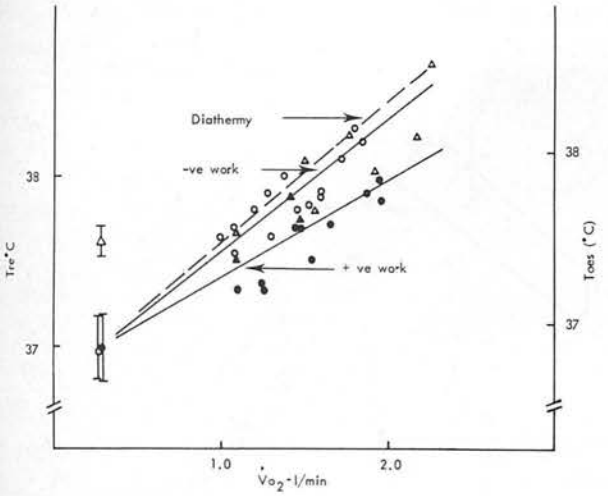

Fig. 1. The relationship between rectal temperature (T_{re}) and oxygen intake ($\dot{V}O_2$) for negative (○) and positive work (●). The formula for the respective regression lines are given in the text. The points for the diathermy (△) and respective control (▲) experiments in terms of oesophageal temperature (T_{oes} – see Methods) are shown on the right hand scale. It will be noted that this scale has been displaced by 0.15° C to account for the difference between T_{re} and T_{oes} (see Methods and Saltin and Hermansen, 1966)

At all levels of exercise T_{re} was higher for a given M in negative than in positive work (Fig. 1) but resting T_{re} were similar. The respective regression equations relating the two variables were given by:

$$T_{re}(°C) = 36.82 + 0.579\ \dot{V}O_2\ (l/min) + ve\ \text{work};\quad r = +0.85\,, \tag{1}$$

$$T_{re}(°C) = 36.74 + 0.790\ \dot{V}O_2\ (l/min) - ve\ \text{work};\quad r = +0.94\,. \tag{2}$$

Comparison of the slopes of the regression lines were significantly different at the 1% level. In the diathermy experiments exercise T_{oes} was on average 0.31° C higher than control values (Table 2) and regression of T_{oes} on $\dot{V}O_2$ was similar to that found for negative work:

$$T_{oes}(°C) = 36.62 + 0.829\ \dot{V}O_2\ (l/min);\quad r = +0.98\,, \tag{3}$$

if account was taken of the accepted (Saltin and Hermansen, 1966) differences between T_{oes} and T_{re} during work (Fig. 1). However, at rest T_{oes} rose to 37.55° C during the period of 1 h when diathermy was applied compared to a value of 37.0° C normally observed without diathermy before the commencement of the control experiments (Table 2). Under normal conditions $\bar{T}_{sk}$ for a given dry bulb temperature (T_{db}) was significantly higher in negative than positive work (Fig. 2).

In order for the comparison shown in Table 1 to be made it should be noted (see Methods) that $\bar{T}_{sk}$ had to be either lower in negative work or raised in positive work by small adjustments to the environmental conditions. In diathermy $\bar{T}_{sk}$ was approx-

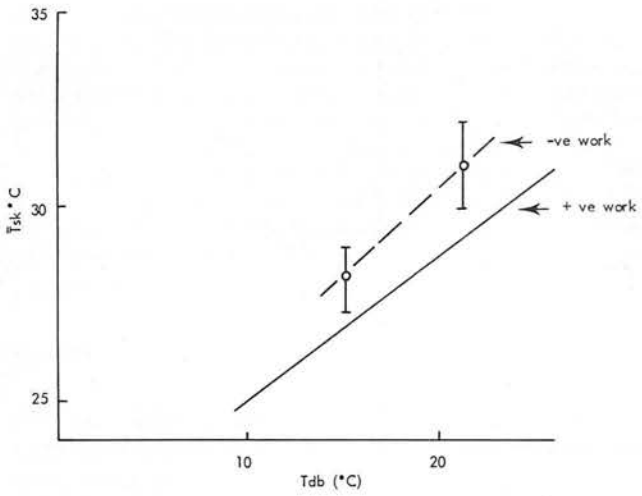

Fig. 2. The relationship between mean skin temperature ($\bar{T}_{sk}$) and dry bulb temperature (T_{db}) during negative work. The full time represents the previously found relationship for positive work (Davies, 1978) under similar convective air flow conditions

imately 29° C during exercise, 1° C higher than we have previously found for positive work at a T_{db} of 21° C ($rh < 50\%$). At the same $\bar{T}_{sk}$, $\dot{M}_{sw}$ was approximately 100 and 173 w/m² higher in negative work and positive work with diathermy, respectively than in control experiments. However, if the extra amount of heat which was absorbed (in the form of work) during negative work and induced during diathermy was taken into account the relationship of $\dot{M}_{sw}$ to H was identical (Fig. 3), in the two forms of exercise and similar to that found in control experiments at the same $\bar{T}_{sk}$. Peripheral tissue heat conductance (K) was higher in negative work and in positive work with diathermy than in normal exercise but at a given $\bar{T}_{sk}$ of approximately 29° C in the former two forms of work the relationship of $\dot{M}_{sw}$ to K was similar in all three forms of work and was represented by the equation:

$$\dot{M}_{sw}(\mathrm{w/m^2}) = -\ 80.2 + 6.08\ K(\mathrm{w/m^2 \cdot °C});\quad r = +\ 0.77\,. \tag{4}$$

In order to assess the influence of core and skin temperatures on the control of $\dot{M}_{sw}$ the equation previously developed (Davies, 1978) of the form:

$$\%\dot{M}_{sw} = -\ 1814 + 44.45\ T_{re}\ (°\mathrm{C}) + 5.90\ \bar{T}_{sk}(°\mathrm{C})\,,$$

where $\%\dot{M}_{sw}$ is equal to $\dot{M}_{sw}$ expressed as a percentage of the maximal evaporative sweat loss ($\dot{M}_{sw\,\max}$), was applied to the diathermy eccentric work and control data. The results are shown in Figure 4.

In positive work with and without diathermy the predicted and observed $\dot{M}_{sw}$ are almost identical but in negative work $\dot{M}_{sw}$ is seriously underestimated particularly at the higher levels of work (Fig. 4). Since $\bar{T}_{sk}$ was identical in negative and positive work (Table 1) the only remaining major thermal input (cf. Nadel et al., 1972) which

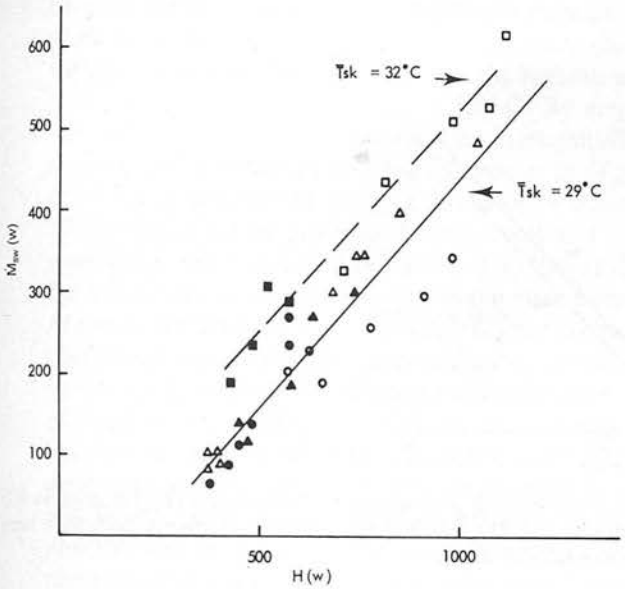

Fig. 3. The relationship of evaporative sweat loss ($\dot{M}_{sw}$) to total heat production (H) during negative work and positive work with and without diathermy at $\bar{T}_{sk}$ of approximately 29° C (symbols as Fig. 1), and positive (■) and negative (□) work at $\bar{T}_{sk}$ of approximately 32° C

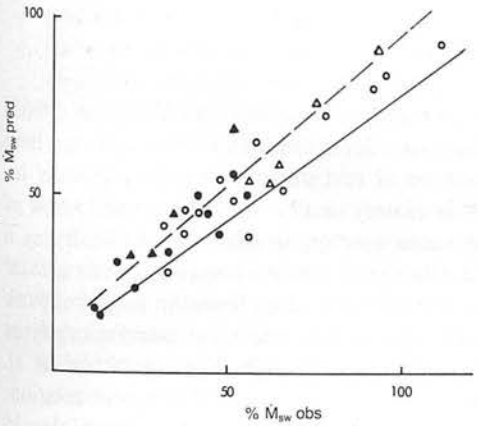

Fig. 1. The relationship of predicted relative sweat rate ($\%\dot{M}_{sw\ pred}$ — see text) to the observed sweat rate ($\dot{M}_{sw\ obs}$) in negative (○) and positive work (●) and in diathermy (△) and control experiments (▲). The regression line (- - -) is given by: $\%\dot{M}_{sw\ pred} = 11.76 + 0.762\ \%\dot{M}_{sw\ obs}$, $r = +0.93$ $\%\dot{M}_{sw\ pred}$ for the negative work observations includes a factor for T_m (see text). The uncorrected relationship for $\%\dot{M}_{sw\ pred}/\%\dot{M}_{sw\ obs}$ in negative is $\%\dot{M}_{sw\ pred} = 10.91 + 0.62\ (\%\dot{M}_{sw\ obs})$, $r = +0.94$ and indicated by the full line

could conceivably influence the results was the temperature of the working muscles (T_m). Figure 5 shows the relationship of T_m to T_{oes} in experiments in which both variables were measured during negative and positive (with and without diathermy) work. For a given T_{oes}, T_m is only marginally higher in positive work with diathermy than in control experiments. Indeed, equally high values of T_m could be obtained at rest with diathermy as those observed in exercise. However, in negative work T_m was consistently higher for given T_{oes} than found in positive control and diathermy exercise experiments. At the highest levels of work the difference in T_m observed in positive and negative work was of the order of 1.6° C (Fig. 5). Assuming that T_m

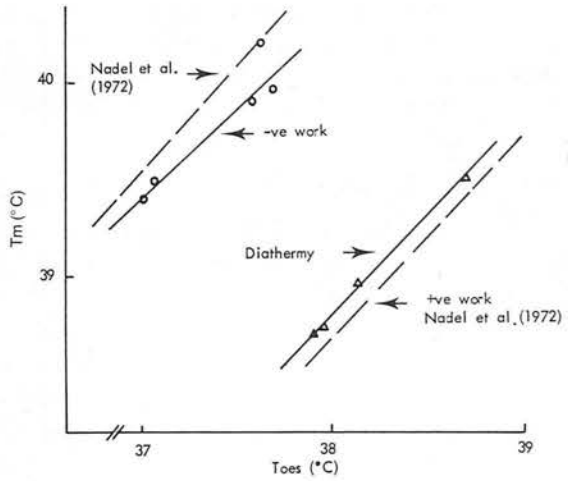

Fig. 5. The relationship of muscle temperature (T_m) to oesophageal temperature (T_{oes}) in negative (O) work and positive work with diathermy (△). The solid lines for negative and positive work have been calculated from the data of Nadel et al. (1972)

accounts for $^1/_{10}$ of the thermal input to sweat in negative work (Stolwijk and Hardy, 1966; Nadel et al., 1972) the differences in sweating response between the three forms of exercise could be resolved (Fig. 4).

Discussion

The results of the effects of diathermy at rest confirm Nielsen and Nielsen's (1965) observation that the core temperature rises to a level commensurate with the heat induced. The mean T_{oes} of 37.60° C observed at rest during the diathermically induced heat load of approximately 290 W is closely similar to the expected value of T_{oes} for the subject when producing the same amount of heat metabolically as a result of exercise (Fig. 1). During exercise with diathermy the rise in T_{oes} was greater than observed during control experiments but similar to that found in negative work (Fig. 1). The increased core temperature for a given M in eccentric exercise confirms our previous observations (Davies and Barnes, 1972) and those of Nadel et al. (1972). In all three forms of exercise at a given $\bar{T}_{sk}$, $\dot{M}_{sw}$ retained constant relationship to H (Fig. 3), though for a given environmental condition $\bar{T}_{sk}$ was higher in negative work (Fig. 2) and in positive work with diathermy than in normal exercise. In order to make comparisons of negative and positive work with and without diathermy at the same $\bar{T}_{sk}$ (Table 1 and 2) it was necessary to modify the environmental conditions. This should be bourne in mind when interpreting the present results.

At a given $\bar{T}_{sk}$ and the same M, $\dot{M}_{sw}$ was significantly higher in negative work and in positive work with diathermy than control experiments (Table 1 and 2). In diathermy this difference in $\dot{M}_{sw}$ could be accounted for by considering T_{oes} but in negative work this factor alone was insufficient to explain the enhanced sweating response (Fig. 4). The differences in sweating response in negative work could only

be resolved by considering an additional thermal input, namely, T_m. In negative work for a given M or T_{oes}, T_m was consistently higher than found in positive work with or without diathermy (Fig. 5). At the highest negative work load the difference in T_m amounted to approximately 1.6° C. By empirically taking account of this increased T_m in terms of a modification in magnitude of the central thermal input to the sweating mechanism then the differences in $\dot{M}_{sw}$ between the different forms of work could be removed. Using a model of sweating previously developed (Davies, 1978) $\dot{M}_{sw}$ could be predicted for the combined diathermy eccentric and control work data with a standard deviation of $\pm$ 10% (Fig. 4). Thus from this view point the differences in core temperature in the three forms of exercise at a given $\bar{T}_{sk}$ and M can be resolved and do not create a barrier to our understanding of thermoregulation during work. The core temperature rises to meet the requirements of heat dissipation by stimulating the sweating mechanism and thereby heat loss, independently of whether heat is induced in the body by diathermy or generated by metabolism or absorbed if the form of work is eccentric exercise. Indeed the thermal responses to diathermy and eccentric work may be regarded as identical (Figs. 1 and 3). I agree with Nadel et al. (1972) that the postulation of specific "work" factors is totally unwarranted by those (see Nielsen, 1969 for general review) who seek to explain thermoregulation during exercise in these terms though of course this does not mean that exercise can be equated directly with the resting state.

At rest the model of sweating proposed for exercise overestimates $\dot{M}_{sw}$ by approximately 20%. This only serves to confirm the potentiating effects of work on sweating which are well known (Robinson et al., 1965; Stolwijk et al., 1968; Smiles and Robinson, 1971). Undoubtedly, the sensitivity of the heat dissipating mechanisms are enhanced by work but nevertheless it would seem that under all conditions of thermal load the central and peripheral temperature responses are better analysed in terms of control and maintenance of heat balance. The rise in core temperature is seen as providing the essential thermal gradient from core to periphery, the central stimulus to sweating, and (if K may be used as an indicator – see Nielsen, 1969) peripheral blood flow (equation IV and see Wyss et al., 1974). Thus cental temperature is maintained during exercise at a level to meet the requirements of heat flow to and from the skin. The rise in core temperature to produce a given relative sweat rate ($\%\dot{M}_{sw}$) at given $\bar{T}_{sk}$ has been found to be constant and independent of the sex and age in individuals who have similar patterns of habitual activity (Davies, unpublished observations). The role of the environment is seen as modifying the constant central output at a given M by its direct action on $\bar{T}_{sk}$, and flow in the peripheral blood vessels. A fall in ambient temperature will cool the skin and inhibit sweating (Stolwijk et al., 1968; Nielsen, 1969; Nadel et al., 1971) and by direct action on superficial veins of the skin increase vasoconstrictor tone (Barcroft and Edholm, 1946; Bevegård and Shepherd, 1967). This would explain the minimal effect of the environment on T_{oes} or T_{re} over a wide range (Nielsen, 1938; Lind, 1963), the temporal relationship of T_{oes} with $\dot{M}_{sw}$, at a given $\bar{T}_{sk}$, during exercise (Nielsen and Nielsen, 1965) and provide a rational basis for the understanding of thermoregulation during exercise in fundamental terms of heat production and dissipation.

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Physiologic Responses to Exercise in Myocardial Infarction Patients Following Residential Rehabilitation

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ABSTRACT. Sargeant AJ, Crawley MA, Davies CTM: Physiologic responses to exercise in myocardial infarction patients following residential rehabilitation. Arch Phys Med Rehabil 60:121-125, 1979.

The physiological responses to exercise were studied in 16 men (33-52 years of age), 8–12 weeks after a first uncomplicated myocardial infarction and following a 3–4 week period of attendance at a residential rehabilitation center at which the patients were required to participate in a controlled program of exercise, sport and recreation. Data were also collected on an inactive, but otherwise healthy group of men of the same age and occupational status, and on an occupationally and recreationally active healthy group. The patient group were indistinguishable from the healthy inactive group in their response to submaximal exercise, although both of these groups showed differences when compared with the active group. Symptom-limited maximal data were also collected and these are reported in relation to the energy requirements of some common leisure, occupational and domestic activities.

The preventative role of physical exercise in coronary heart disease has been examined by several workers in recent years. In particular epidemiological studies have demonstrated an apparent association between occupational activity level, and more recently vigorous leisure time activity, and the incidence of coronary heart disease.[1-5] The role of exercise once the disease has become manifest is less certain although large scale ongoing investigations in Sweden[6] and elsewhere are exploring this question.

Notwithstanding the uncertainties which remain, it is clear that medical practice should aim to rehabilitate both physically and psychologically the patient who has suffered a myocardial infarction so that he is able to function with confidence within his own limitations. To attain this objective a graded program of exercise therapy and testing under controlled clinical conditions is desirable so that both patient and physician have an informed understanding of the limitations and problems associated with physical exercise.

A period of residential rehabilitation as previously described[7] provides a good opportunity for this type of work, and within the British Armed Services this facility is provided by the Royal Air Force Medical Rehabilitation Unit (RAF MRU). This paper illustrates the application of this approach and presents data collected on patients attending RAF MRU after a first uncomplicated myocardial infarction. These data are compared with those in 2 healthy groups of men, one inactive and the other active.

Subjects

The patients were 16 senior noncommissioned and commissioned male officers (age range 33–52 years) who had suffered a first uncomplicated myocardial infarction. All were occupationally and recreationally inactive prior to the infarction. They were seen during attendance at RAF MRU for a 3–4 week period of residential rehabilitation therapy. The measurements reported here were made at the time of discharge from the unit which was from 8–12 weeks postinfarction. On the basis of their response to exercise, patients were advised to take regular walking and/or jogging exercise of from 15–30 minutes duration daily 3–4 days per week after discharge. The measurements were repeated on the patients 4 months later, during which time they had returned to their jobs.

Data were also collected on 2 groups of healthy men: the first of these was composed of 16 men of the same occupation and age as the patients (referred to as the inactive normal group); the second was a group of 10 middle-aged service personnel who were occupationally and recreationally very active (physical training instructors who were qualified remedial gymnasts, referred to as the active normal group).

All subjects, both patients and healthy persons, gave their informed consent to take part in the study which was carried out under the auspices of the Army Personnel Research Committee of the Medical Research Council.

Methods

The subjects were studied in the morning 2–3 hours after a light breakfast. They were examined at rest by the responsible medical officer, with blood pressure and electrocardiographic (ecg) data recorded with the subjects in the supine position.

Subjects were then seated on an electrically braked bicycle ergometer[a] and performed a continuous progressive exercise test, starting at zero load and increasing by 50 watts every 4 minutes. The end point was determined either by the subject becoming exhausted and unable to continue (subject limited) or in

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the case of 6 patients, by intervention of the medical officer (observer limited) following the appearance of symptoms contraindicating further exercise.[8]

Cardiorespiratory measurements were made over the last 2 minutes of each work load. An open circuit technique[9] was used to measure minute ventilation ($\dot{V}_E$), oxygen uptake ($\dot{V}O_2$) and carbon dioxide output ($\dot{V}CO_2$). The ecg was displayed continuously on an oscilloscope and recorded throughout exercise and during the recovery period. Blood pressure was measured using a mercury sphygmomanometer prior to exercise (supine) and after 1 minute of exercise at each work load.

The submaximal response to exercise was characterised by fitting linear regressions to the individual data and predicting the response at a standard value of the independent variable.[10] Hence minute ventilation has been predicted at a carbon dioxide output of 1.5 liters/min ($\dot{V}_{E\ 1.5}$); cardiac frequency at an oxygen uptake of 1.5 liters/min ($f_{H\ 1.5}$); oxygen uptake at a cardiac frequency of 185 beats/min ($\dot{V}O_{2\ 185}$) and at a work load of 150 watts ($\dot{V}O_{2\ 150}$); and tidal volume at a minute ventilation of 30 liters/min ($V_{T\ 30}$).

After the subjects had fully recovered from the exercise test, their weight, height and lean body mass (estimated from the sum of 4 skinfold thicknesses[11]) were measured.

Results

There were no significant differences among the 3 groups in either mean age (43 years) or their physical characteristics (height 174 cm; weight 73.7 kg; lean body mass 61.7 kg). Lean body mass indicated that 17% of the body weight of the cardiac patients and inactive normal subjects was attributable to fat content and this was only slightly and not significantly greater than the value (15%) found in the active group. Remeasurement of the patients after 4 months demonstrated no significant changes.

Resting (supine) cardiac frequency was slightly but not significantly lower in the active group (63 ±10 beat/min) compared with the patient and inactive groups (73 ±9 and 70 ±10 beats/min, respectively). There were no significant differences among the groups in resting (supine) systolic or diastolic blood pressures which were 124/84, 126/82 and 130/83 mm Hg in the patient, inactive and active groups, respectively.

SUBMAXIMAL RESPONSES TO EXERCISE

The means (±SD) of the submaximal response to exercise are summarized in table 1.

No significant differences were found between the cardiac patients and the inactive healthy subjects. However in the active subjects, $f_{H\ 1.5}$ was significantly ($p < 0.01$) reduced by about 20 beats/min when compared with both of these groups and this difference was also reflected in $\dot{V}O_{2\ 185}$ which was significantly greater (+ 30%; $p < 0.01$) in the active group.

The only significant change observed in these responses when the patients were remeasured 4 months after discharge was an increase in $V_{T\ 30}$ of 0.16 liters ($p < 0.01$).

There were no significant differences among the 3 groups in the systolic and diastolic blood pressure in response to increasing work load.

The systolic blood pressure/cardiac frequency product was significantly lower in the active group at a given work load when compared with the inactive group and the patients, but there was no significant difference between the latter groups (fig 1).

MAXIMAL RESPONSES TO EXERCISE

All of the healthy subjects and 10 of the cardiac patients were allowed to continue exercise until they felt unable to continue due to exhaustion (subject

Table 1: Submaximal Responses to Exercise in Three Study Groups and Mean Change in Patients Observed 4 Months After Discharge

Group	$\dot{V}_{E\ 1,5}$, liters/min	$f_{H\ 1.5}$, beats/min	$\dot{V}O2\ 185$, liters/min	$\dot{V}O_2\ 150$, liters/min	$V_{T\ 30}$, liters/min
(a) Patients (n = 16)	55.1 ±8.1	135 ±16	2.50 ±0.41	1.92 ±0.09	1.59 ±0.43
(b) Inactive (n = 16)	56.0 ±10.7	132 ±12	2.60 ±0.39	1.94 ±0.18	1.50 ±0.21
(c) Active (n = 10)	53.2 ±6.1	115 a** ±15 b***	3.32 a** ±0.72 b**	2.04 ±0.17	1.53 ±0.24
(d) Patients, 4 mo after discharge (n = 11)	-2.6 (ns)	-4 (ns)	-0.04 (ns)	+0.02 (ns)	+0.16 ($p < 0.01$)

Statistical comparison between groups a, b, and c, *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$. In the case of (d) the significance of the change (paired 't') is given: ns not significant. Minute ventilation at $\dot{V}CO_2$ of 1.5 liters/min ($\dot{V}_{E\ 1.5}$); cardiac frequency at $\dot{V}O_2$ of 1.5 liters/min ($f_{H\ 1.5}$) = oxygen uptake at $f_{H\ 185}$ beats/min and W of 150 watts ($\dot{V}O_{2\ 185}$ and $\dot{V}O_{2\ 150}$); tidal volume at $\dot{V}_E$ liters/min ($V_{T\ 30}$).

Values for (a), (b) and (c) are presented as mean ± standard deviation and for (d) mean change.

Consultation of standard tables of energy expenditure in the light of the patients' exercise performance enables some clear recommendations to be made, although this may be of greatest significance in those patients whose exercise test was observer limited (fig 2).

Finally it is worth emphasizing that there may be important psychological benefits to a rehabilitation program instituted at an early stage following myocardial infarction. We were unable to examine this aspect but clearly it is an important area which deserves further study.

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progressing in many cases from small muscle group activity to running and other whole body vigorous activity. The data reported in this paper were collected at the end of this period of residential rehabilitation immediately prior to discharge.

The characterization of the response to submaximal exercise (table 1) demonstrates no significant differences between the patient and the inactive normal groups. In contrast the active group have a marked decrease in cardiac frequency at a given oxygen uptake of 1.5 liters/min when compared with either the patients or inactive groups (115 as compared to 135 and 132 beats/min, respectively). This is a well recognized effect of increased activity level and implies an approximately 10% higher exercise stroke volume in the active group, if cardiac output for a given $\dot{V}O_2$ is maintained.[17] This difference is also reflected in the lower ($\sim$10 beats/min) resting cardiac freuqency of the active group.

The product of systolic blood pressure (SBP) and cardiac frequency (f_H) shows a similar linear relationship with increasing work load in both the patient and inactive groups, while the levels observed in the active group are somewhat lower at a given work load (fig 1). In healthy subjects it has been suggested[18,19] that the product of SBP x f_H reflects myocardial blood flow and hence if comparison between the groups is valid it would suggest that as in other submaximal exercise responses the patients behave in a similar way to the inactive healthy subjects. The lower values of SBP x f_H and presumably myocardial blood flow in the active group may be due to a reduction in myocardial oxygen requirements at a given cardiac output resulting from an increase in myocardial efficiency associated with a larger stroke volume.[20]

Predicting oxygen uptake at the population mean value of maximal cardiac frequency for this age group (that is, 185 beats/min)[21] again demonstrates no significant difference between the patients and the inactive subjects (2.5 ±0.41 and 2.6 ±0.39, respectively) although $\dot{V}O_{2\ 185}$ in the active group is significantly higher (3.32 ± 0.72 liters/min).

Hence despite having had a myocardial infarction, the responses of the patient group to submaximal exercise were not demonstrably different from those of a group of apparently healthy but inactive subjects from the same occupational and age group. However, whether the inactive healthy subjects demonstrate desirable responses to exercise may be debatable; a more significant comparison may be made with the active subjects from whom both the patient and inactive groups differ in many respects.

Ten patients were allowed to continue exercise to exhaustion (subject limited maximum - SL max). $\dot{W}_{SL\ max}$ and $\dot{V}O_{2\ SL\ max}$ attained by the cardiac patients are significantly lower (14%; $p < 0.01$) than those of the normal inactive subjects, and $f_{H\ SL\ max}$ is also lower by 11 beats/min although not significantly (table 2). In the absence of definitive maximal data it is not possible to decide whether these are genuine physiological differences in maximal function or whether they are simply due to persistent and understandable caution on the part of the patients regarding vigorous exercise. Not surprisingly the active group show higher SL_{max} levels of $\dot{W}$, $\dot{V}_E$ and $\dot{V}O_2$, than either the patients or inactive subjects, underlining the improved cardiovascular function identified in the submaximal responses.

Notwithstanding the problem of the physiological interpretation of the subject limited maximum it is a practical measure of the level to which the patient will voluntarily go without untoward symptoms. In this sense it is a useful basis on which to make recommendations regarding desirable levels of activity following discharge from direct care. Figure 2 gives the individual value for the normal subjects and patients against typical energy expenditure values taken from the literature for a variety of common activities.[22] Some care is needed in the interpretation of these values which are based on a 75 kg man, since account must often be taken of body weight as well as of variations in the nature of the activities themselves.

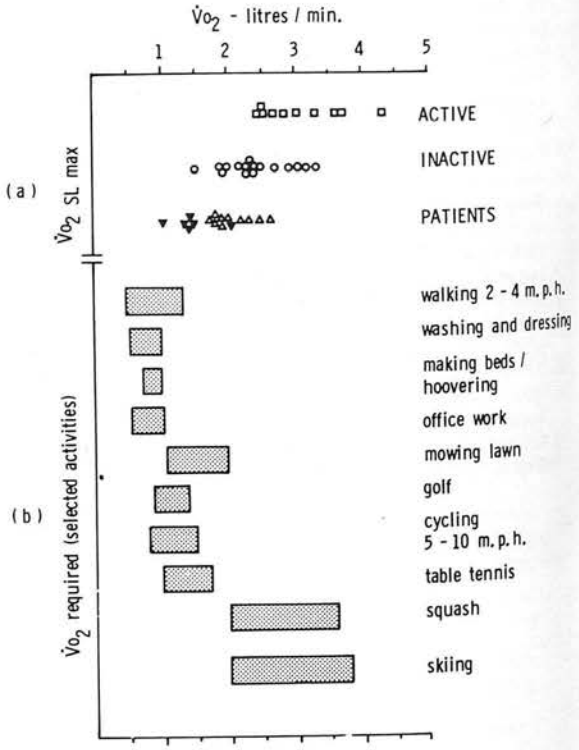

Fig 2—(a) Individual values for subject-limited maximal oxygen uptake ($\dot{V}O_{2\ SL\ max}$) achieved by the active (□) and inactive (○) normal subjects and patients (△). Also shown are the levels achieved in 6 cases where exercise was limited by the observer (▼). (b) Examples of the range of oxygen uptake required in some everyday domestic, occupational and leisure activities (from Durnin and Passmore[22]).

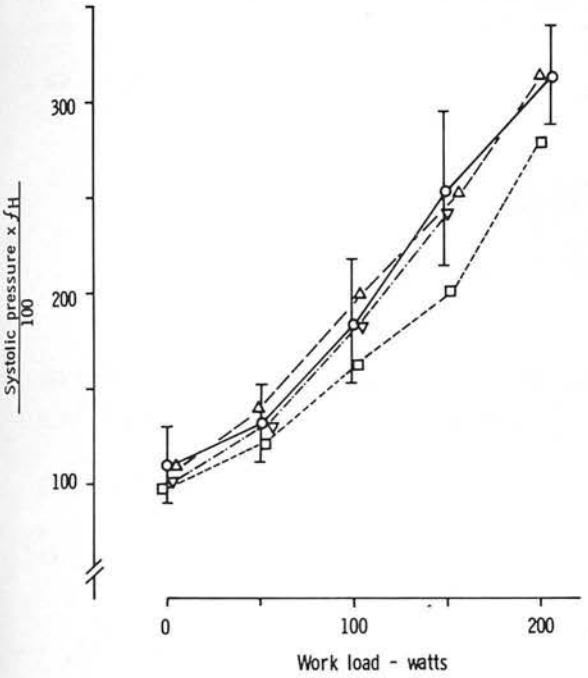

Fig 1 – Systolic blood pressure x cardiac frequency product (arbitrary units) in relation to work load. The standard deviation for the normal inactive (○) data is shown along with mean values for the active group (□) and patient groups at the time of discharge (△) and 4 months later (▽).

limited maximum). These data are summarized in table 2.

The maximum (subject limited) work load ($\dot{W}_{SL\ max}$) and oxygen uptake ($\dot{V}O_{2\ SL\ max}$) achieved by the patient group were significantly lower (−14%; $p < 0.01$) than the levels reached by the inactive subjects. Similar, but nonsignificant reductions are shown in maximal ventialtion ($\dot{V}_{E\ SL\ max}$) and cardiac frequency ($f_{H\ SL\ max}$).

$\dot{W}_{SL\ max}$, $\dot{V}_{E\ SL\ max}$ and $\dot{V}O_{2\ SL\ max}$ achieved by the active group were significantly larger than those in both the patient and inactive groups, exceeding the latter group's values by about 25%. In both the active and inactive groups $f_{H\ SL\ max}$ was the same (176 beats/min).

There were no significant changes in $\dot{W}_{SL\ max}$, $\dot{V}O_{2\ SL\ max}$ or $f_{H\ SL\ max}$ attained by 5 patients who were remeasured 4 months after discharge although $\dot{V}_{E\ SL\ max}$ had significantly ($p < 0.05$) declined by 10 liter/min.

Discussion

Physical training may have an important physiological and psychological role to play in the treatment of many patients following myocardial infarction,[6, 12-15] and it has been demonstrated that patients following an extended program of outpatient treatment can eventually reach high levels of physical fitness.[16] However, for many patients this type of long-term training program is impossible to sustain, resulting in a high drop-out rate.[6] It could be argued therefore that greater priority should be given to the early management of the disease, incorporating assessment of the physiological response to exercise under clinical conditions. Thus the patient may be quickly restored to an active life with a confident understanding of his functional capacity and with clear objectively based advice from his physician on desirable levels of exercise.

It is with these aims in mind that suitable patients are offered the opportunity of attending for a 3 to 4 week period of residential rehabilitation at RAF MRU within 8 weeks of the myocardial infarction. On arrival at RAF MRU, patients were still very cautious regarding exercise and were effectively still at a relatively early phase of mobilization. Accordingly and dependent upon regular clinical assessment, including exercise testing, patients were given a carefully graded program of exercise and recreationally based therapy,

Table 2: Mean (±SD) of the Subject Limited Maximal Responses in Three Study Groups and Mean Change in Patients Observed 4 Months after Discharge

Group	$\dot{W}_{SL\ max}$, watts	$\dot{V}_{E\ SL\ max}$, liters/min	$\dot{V}O_{2\ SL\ max}$, liters/min	$f_{H\ SL\ max}$, beats/min
(a) Patients (n = 10)	170 b** ±26	80.8 ±13.3	2.10 b** ±0.30	165 ±14
(b) Inactive (n = 16)	197 ±39	97.3 ±29.6	2.45 ±0.51	176 ±17
(c) Active (n = 10)	250 a*** ±41 b**	126.6 a*** ±15.3 b**	3.09 a*** ±0,64 b**	176 ±12
(d) Patients, 4 mo after discharge (n = 5)	0 (ns)	-10.4 ($p < 0.05$)	-0.03 (ns)	-4 (ns)

Statistical comparisons as for table 1. Work load ($\dot{W}_{SL\ max}$); minute ventilation ($\dot{V}_{E\ SL\ max}$); oxygen uptake ($\dot{V}O_{2\ SL\ max}$); and cardiac frequency ($f_{H\ SL\ max}$).

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Aerobic Performance of Female Marathon and Male Ultramarathon Athletes

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Summary. The aerobic performance of thirteen male ultramarathon and nine female marathon runners were studied in the laboratory and their results were related to their times in events ranging in distance from 5 km to 84.64 km.

The mean maximal aerobic power output ($\dot{V}O_{2\ max}$) of the men was 72.5 ml/kg · min compared with 58.2 ml/kg · min ($p < 0.001$) in the women but the O_2 cost ($\dot{V}O_2$) for a given speed or distance of running was the same in both sexes. The 5 km time of the male athletes was closely related to their $\dot{V}O_{2\ max}$ ($r = -0.85$) during uphill running but was independent of relative power output ($\%\dot{V}O_{2\ max}$). However, with increasing distance the association of $\dot{V}O_{2\ max}$ with male athletic performance diminished (but nevertheless remained significant even at 84.64 km), and the relationship between $\%\dot{V}O_{2\ max}$ and time increased. Thus, using multiple regression analysis of the form:

42.2 km (marathon) time (h) = 7.445 − 0.0338 $\dot{V}O_{2\ max}$ (ml/kg · min) − 0.0303% $\dot{V}O_{2\ max}$ ($r = 0.993$)

and

84.64 km (London–Brighton) time (h) = 16.998 − 0.0735 $\dot{V}O_{2\ max}$ (ml/kg · min) − 0.0844% $\dot{V}O_{2\ max}$ ($r = 0.996$)

approximately 98% of the total variance of performance times could be accounted for in the marathon and ultramarathon events. This suggests that other factors such as footwear, clothing, and running technique (Costill, 1972) play a relatively minor role in this group of male distance runners. In the female athletes the intermediate times were not available and they did not compete beyond 42.2 km (marathon) distance but for this event a similar association though less in magnitude was found with $\dot{V}O_{2\ max}$ ($r = -0.43$) and $\%\dot{V}O_{2\ max}$ ($= -0.49$). The male athletes were able to sustain 82% $\dot{V}O_{2\ max}$ (range 80–87%) in 42.2 km and 67% $\dot{V}O_{2\ max}$ (range 53–76%) in 84.64 km event. The comparable

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figure for the girls in the marathon was 79% $\dot{V}O_{2\ max}$ (ranges 68–86%). Our data suggests that success at the marathon and ultramarathon distances is crucially and (possibly) solely dependent on the development and utilisation of a large $\dot{V}O_{2\ max}$.

Key words: Aerobic performance – Athletes, marathon, ultramarathon

The physiology of male marathon running has been studied extensively in the laboratory (Costill and Fox, 1969; Costill, 1970, 1972; Costill et al., 1971; Pollock, 1977) and in the field (Pugh et al., 1967; Magazanick et al., 1974; Maron et al., 1976, 1977) but there are few data on women (Wilmore and Brown, 1974; Daniels et al., 1977). However, at distances beyond the traditional marathon (42.2 km) event there have been no systematic observations made on athletes of either sex. Costill and Winrow (1970) reported the metabolic cost of running for 2.5–5.5 h on two middle-aged distance athletes and Dancaster and Whereat (1971) observed fluid balance changes during the comrades (86.9 km) marathon race, but the physiology of ultralong-distance running has not been investigated. The present study is concerned with aerobic performance in the laboratory and field of British male ultralong-distance and female marathon athletes. The girls were nationally ranked athletes and the men contained the world record holder for the 50 (80.48 km) and 100 (160.93 km) miles and the 3rd ranked athlete at 24 h distance running.

Subjects and Methods

The physical characteristics of the male and female athletes studied are given in Table 1. All the male athletes regularly competed in marathon and ultramarathon races. In addition to CW (world record holder) TO'R was an outstanding ultralong-distance runner having won the prestigous London to Brighton race (84.64 km) in 1976 in 5 h 23 min. TR was the current (1977) Road Runners Club 24 h event champion covering 251.46 km. At the time of testing in the laboratory, all male athletes were in training (120–250 km per week). The best female athlete was LB who had a time for the marathon of 2 h 57 min, though PD and BN were outstanding older ♀ athletes. Like the men they were all in regular training when measured, though their weekly training schedules were less arduous (80–100 km per week).

The oxygen cost of running on the treadmill at various speeds was determined for all athletes using an open circuit technique, collection being made through a modified, low resistance, Otis-McKerrow valva and 1.5″ smooth bore tubing into a conventional Tissot spirometer. The O_2 and CO_2 content of

Table 1. Physical characteristics and maximal aerobic power output ($\dot{V}O_{2\ max}$) of the 13 male and 9 female athletes (Mean ± SD)

	Age (year)	Wt (kg)	Ht (cm)	$\dot{V}_{E\ max}$ l/min	$\dot{V}O_{2\ max}$ l/min	ml/kg · min	$f_{H\ max}$ beats/min
♂	33.0 ± 6.3	63.5 ± 6.9	172.3 ± 8.1	162.5 ± 19.4	4.64 ± 0.48	72.5 ± 3.8	194 ± 10
♀	29.8 ± 7.8[c]	52.3 ± 2.6[a]	162.5 ± 2.2[a]	115.2 ± 12.8[a]	3.04 ± 0.19[a]	58.2 ± 4.8[a]	197 ± 6

♂ ♀ Significance: [a] $p < 0.001$; [b] $p < 0.01$; [c] $p < 0.05$

the expired air was determined by physical gas analysers (Beckman and Servomex Instruments Ltd.) which were regularly calibrated with gases of known concentrations from prior (Lloyd) Haldane chemical analysis. The maximal oxygen intake ($\dot{V}O_{2\ max}$) was either measured on the same or a subsequent occasion (Table 1) using the treadmill and the criteria outlined by Davies (1968). The performance times of the athletes on the track and in competitive road running were obtained by questionnaires and from the official records of the Road Running Club of Great Britain.

Results

The male athletes were heavier, taller, less fat and had higher aerobic power outputs than their female counterparts (Table 1), but despite these differences the O_2 cost of running per kg body weight ($\dot{V}O_2$ ml/kg · min) at various speeds was the same in both groups (Fig. 1). There were no significant differences between either the slopes or the intercepts of the male and female regression lines ($p > 0.1$). The combined regression for the ♂ and ♀ athletes was given by:

$$\dot{V}O_2\ (\text{ml/kg} \cdot \text{min}) = -7.736 + 3.966\ \text{speed (km/h)};\ (r = +0.94)\ (n = 77). \quad (1)$$

The mean $\dot{V}O_{2\ max}$ in the males was 72.5 (± 3.8) ml/kg · min compared with 58.2 (± 4.8) ml/kg · min in the females. The best guide to $\dot{V}O_{2\ max}$ in the males from their

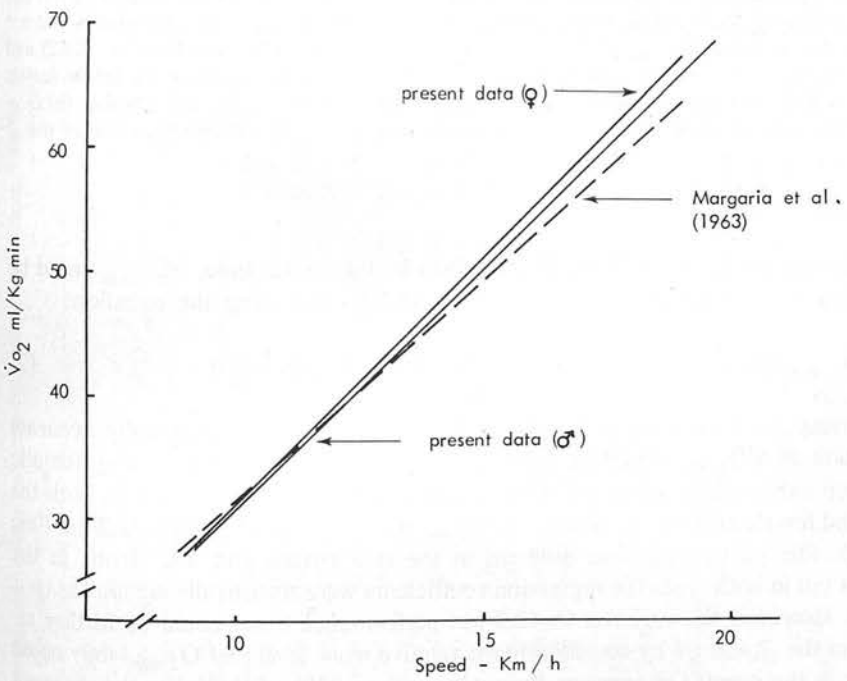

Fig. 1. Oxygen intake ($\dot{V}O_2$) in relation to speed of running at zero gradient on a motor-driven treadmill. Full lines represent the male and female regression lines which are given by: $\dot{V}O_2$ (ml/kg · min) = 8.393 + 3.998 speed (km/h) and $\dot{V}O_2$ (ml/kg · min) = 8.589 + 4.046 speed (km/h), respectively. The dotted line is taken from Margaria et al. (1963)

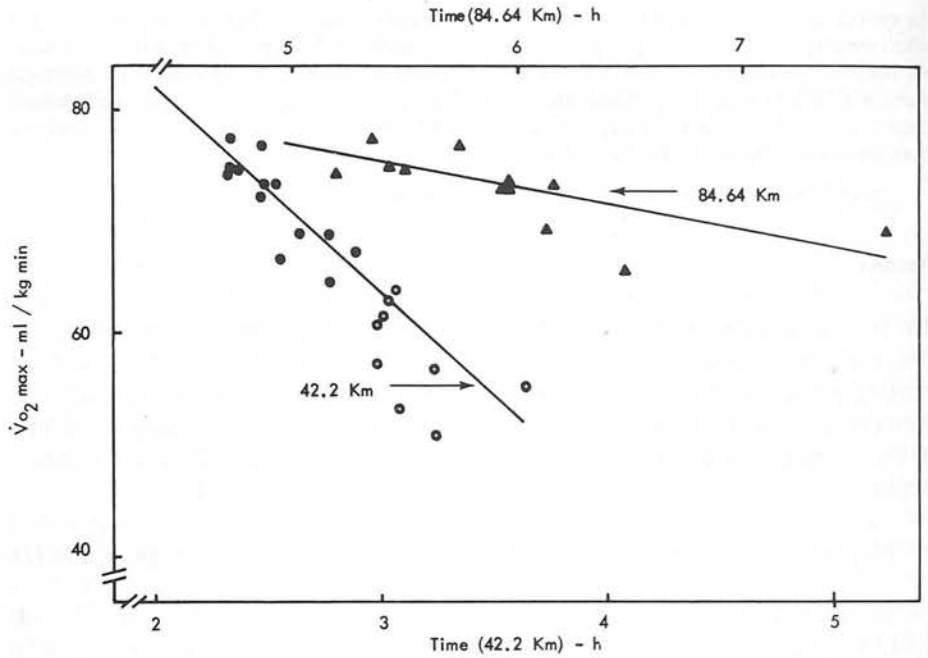

Fig. 2. Maximal aerobic power output ($\dot{V}O_{2\ max}$) in relation to the marathon (42.2 km) and the ultra-marathon (84.64 km London–Brighton) times in the male and female athletes. Symbols: 42.2 km (●) males, (○) females. 84.64 km (▲) males. The regression lines are shown for the male athletes and are given by the equations: $\dot{V}O_{2\ max}$ (ml/kg · min) = 117.96 − 18.10. 42.2 km time (h) ($r = -0.82$) and $\dot{V}O_{2\ max}$ (ml/kg · min) = 96.18 − 3.88. 84.64 km time (h) ($r = -0.72$). The regression line for the female athletes at 42.2 km is represented by $\dot{V}O_{2\ max}$ (ml/kg · min) = 87.69 − 9.35. 42.2 km time (h) ($r = -0.43$). The slope of the regression equation is significantly ($p < 0.05$) different from that of the ♂ athletes

performance on the track (Table 2) was given by their 5 km time, $\dot{V}O_{2\ max}$ could be predicted with a standard deviation of ± 2 ml/kg · min using the equation:

$$\dot{V}O_{2\ max}\ (\text{ml/kg} \cdot \text{min}) = 129.73 - 3.617.\ 5\ \text{km time (min)}. \quad (2)$$

Converting the 5 km time to speed (km/h) and using Eq. (1) an equally accurate prediction of $\dot{V}O_{2\ max}$ could be made but the procedure resulted in a systematic underestimation of the observed value of approximately 6% (Table 2). In both the male and female athletes the observed $\dot{V}O_{2\ max}$ was also related to their 42.2 km time (Fig. 2). The relationship was different in the two groups and less strong in the females but in both cases the regression coefficients were statistically significant ($p < 0.001$). However, the variance in 42.2 km performance times could be further reduced in the ♂ and ♀s by considering the relative work level (%$\dot{V}O_{2\ max}$) they could sustain in the event. On average, the males utilised 82% of their directly observed $\dot{V}O_{2\ max}$ (and sustained a speed equal to 89% of that attained at 5 km − Table 2) compared with 79% in the female athletes (Table 3) but again in both groups the %$\dot{V}O_{2\ max}$ was critically related to performance (Fig. 3). In contrast to the relation-

Table 2. Aerobic and running performance in male athletes from 5 km to 84.64 km. Time, speed, oxygen intake ($\dot{V}O_2$) estimated from Equation 1 (see text) and relative work load ($\%\dot{V}O_{2\,max}$)

Athlete	5 km				42.2 km					84.64 km				
	Time (min)	Speed (km/h)	$\dot{V}O_2$ (ml/kg · min)	$\%\dot{V}O_{2\,max}$ obs.	Time (h)	Speed (km/h)	%[a]	$\dot{V}O_2$ (ml/kg · min)	$\%\dot{V}O_{2\,max}$ obs.	Time (h)	Speed (km/h)	%[a]	$\dot{V}O_2$ (ml/kg · min)	$\%\dot{V}O_{2\,max}$ obs.
TK	14.48	20.72	74.4	100.0	2.315	18.23	88.0	64.6	86.8	5.503	15.38	74.2	53.3	71.6
IB	14.88	20.16	72.2	96.7	2.376	17.76	88.0	62.7	83.9	5.442	15.55	77.1	53.9	72.2
JO	15.48	19.38	69.1	93.8	2.495	16.91	87.3	59.3	80.5	6.192	13.67	70.5	46.5	62.2
AJ	17.62	17.03	59.8	90.7	2.888	14.61	85.8	52.2	79.2	6.483	13.06	76.7	44.1	66.9
MWT	16.13	18.59	66.0	90.0	2.469	17.09	91.9	60.0	82.6	5.911	14.32	77.0	49.0	67.5
CW	15.02	19.97	71.5	95.2	2.330	18.11	90.7	64.1	85.4	5.202	16.27	81.5	56.8	75.6
TR	15.67	19.17	68.2	88.5	2.475	17.05	88.9	59.9	77.7	5.753	14.71	76.7	50.6	65.6
TO'R	15.07	19.91	71.2	91.4	2.334	18.08	90.8	64.0	82.2	5.392	15.69	78.8	54.5	70.0
TP	17.07	17.57	61.9	92.9	2.561	16.48	93.8	57.6	86.5	–	–	–	–	–
KW	16.10	18.63	66.2	96.1	2.780	15.18	81.4	52.5	76.2	7.637	11.08	59.5	36.2	52.5
DF	16.50	18.18	64.4	93.0	2.618	16.12	88.7	56.2	81.2	6.139	13.79	75.9	46.9	67.8
AJO	15.90	18.87	67.1	90.9	2.512	16.80	89.0	58.9	79.8	5.950	14.23	75.4	48.7	66.0
DB	15.68	19.13	68.1	96.2	2.472	17.07	89.3	60.0	82.2	5.929	14.28	74.6	48.9	67.0
Mean ± SD	15.82 ±0.89	19.02 ±1.05	67.7 ±4.2	93.6 ±3.2	2.510 ±0.172	16.88 ±1.10	88.7 ±3.0	59.4 ±4.0	81.9 ±3.2	5.961 ±0.646	14.34 ±1.39	74.8 ±5.5	49.1 ±5.5	67.1 ±5.8

[a] Speed expressed as % of the 5 km value

Table 3. Aerobic and running performance at 42.2 km in female athletes. Time, speed, oxygen intake ($\dot{V}O_2$) and relative work load ($\%\dot{V}O_{2\,max}$)

Athlete	Time	Speed	$\dot{V}O_2$	$\%\dot{V}O_{2\,max}$
LT	3.069	13.75	46.8	73.7
IW	3.237	13.04	44.0	77.8
LW	3.040	13.88	47.3	76.4
CR	2.979	14.16	48.4	85.3
LB	2.954	14.29	48.9	79.8
PD	3.089	13.66	46.4	87.4
RC	3.037	13.90	47.4	73.6
BN	3.242	13.02	43.9	86.4
JD	3.667	11.51	37.9	68.0
Mean	3.146	13.47	45.7	78.7
± SD	±0.219	± 0.85	± 3.4	± 6.6

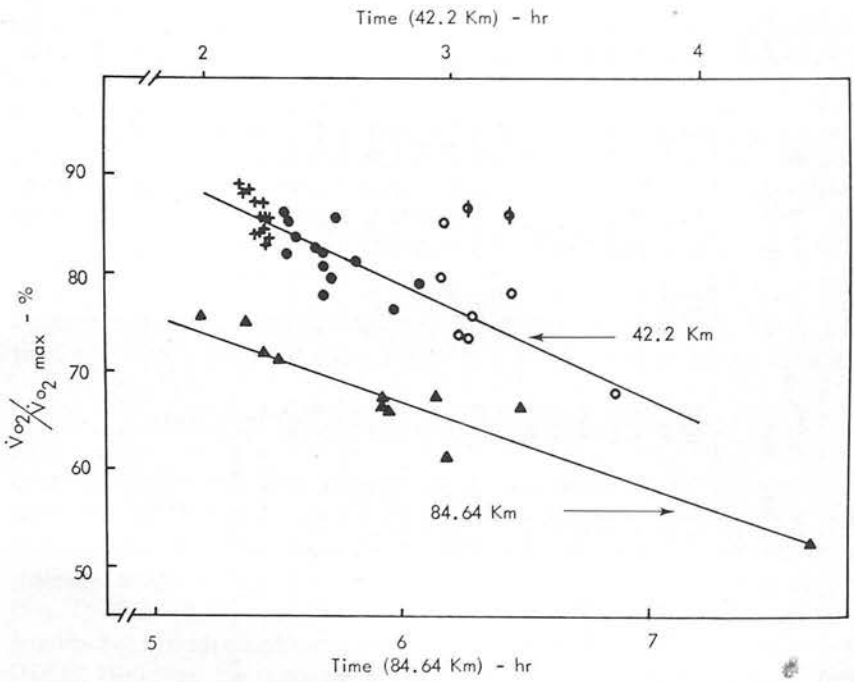

Fig. 3. Relative ($\dot{V}O_{2\,max}$) aerobic energy expenditure in the female marathon (42.2 km) and the male ultramarathon (London to Brighton = 84.64 km) events. The regression (full) lines are given for the males by: $\%\dot{V}O_{2\,max} = 111.52 - 11.82$. 42.2 km time (h) ($r = -0.63$), $\%\dot{V}O_{2\,max} = 116.51 - 8.29$. 84.64 km time (h) ($r = -0.93$). The regression equation for the female athletes at 42.2 km is $\%\dot{V}O_{2\,max} = 125.93 - 14.98$. 42.2 km time (h) ($r = -0.47$) is not statistically different ($p > 0.05$) from the male equation and therefore if one wishes the data may be combined. Symbols: (+) Elite athletes see Table 4; (ϕ) PD and bN see Subjects and Methods and remaining symbols see Fig. 2

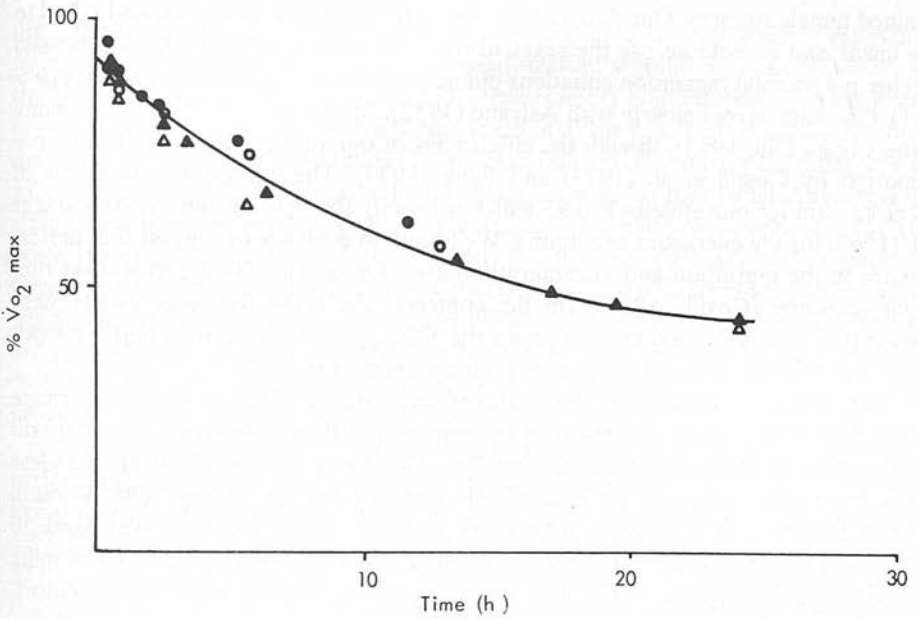

Fig. 4. The relationship of sustainable relative aerobic power output (%$\dot{V}O_{2\ max}$) in relation to time in four elite ultralong-distance athletes. CW (●); TR (△); MWT (○) and DF (▲). The curved line is represented by the equation: %$\dot{V}O_{2\ max}$ = 91.24 − 3.79 time (h) + 0.08 time^2 (h) (r = 0.985)

ship for $\dot{V}O_{2\ max}$ the regression equations of %$\dot{V}O_{2\ max}$ on 42.2 km time could be combined:

$$\%\dot{V}O_{2\ max} = 100.11 - 7.046.\ 42.2 \text{ km time (h)} \quad (r = -0.52).$$

At distances beyond the marathon in the male athletes the relationships of aerobic power to running performance were maintained (Figs. 2 and 3) but at 84.64 km importance of the relative work that could be sustained was increased (r = −0.93) and had a greater influence on performance time than the absolute $\dot{V}O_{2\ max}$ (r = −0.72).

The relationship between time of performance and relative sustainable work level is shown in Figure 4.

Discussion

The results of the present study show that, despite the well defined differences in body size and shape (Table 1) and the recently established (Nelson et al., 1977) biomechanical variation in running techniques between the sexes, the O_2 cost of running on a motor driven treadmill when standardised for body weight is the same in male and female athletes. This observation is at variance with the recent data of Bransford and Howley (1977) who found male athletes to be more economical than

trained female runners. Our data clearly shows the relationship of $\dot{V}O_2$ and speed to be linear and to hold across the sexes in treadmill running (Fig. 1). Quadratic and higher polynomial regression equations did not significantly reduce the variance ($p > 0.1$). Our data agrees closely with Åstrand (1952), Margaria et al. (1963), and many others (e.g., Dill, 1965), though the efficiencies of our runners were less than those reported by Costill et al. (1973) and Pugh (1970). The energy cost in terms of kcal/kg · km for our athletes is 0.95 which is exactly the figure given by Margaria et al. (1963) for the energetics of running. We found no evidence to suggest that performance in the marathon and ultramarathon events was critically dependent on running economy (Costill, 1972). On the contrary, the times for these events were associated with only two major factors the $\dot{V}O_{2\ max}$ and the relative aerobic work level which could be sustained during competition (Fig. 3).

Our $\dot{V}O_{2\ max}$ values for the male athletes (mean 72.5 ml/kg · min; range 65.9–77.9 ml/kg · min) are closely in agreement with those reported for male marathon runners (Costill, 1970; Costill and Fox, 1971) and are 10–12 ml/kg · min less than the figures obtained on elite middle distance runners (Saltin and Åstrand, 1967). However, it is perhaps noteworthy that our athlete DF who was aged 50 years and is the subject of further series of observations concerned with 24 h running (Davies and Thompson, 1979), had a $\dot{V}O_{2\ max}$ of 69.2 ml/kg · min. To our knowledge this is the highest value of aerobic power output reported for a man of his age. It may be compared to Costill's subject Cor's (aged 49 years – 42.2 km time 2.691 h) value of 65.1 ml/kg · min and the famous marathon runner De Mar who had at the age of 49 years a $\dot{V}O_{2\ max}$ of 60 ml/kg · min (Dill, 1965). The women athletes mean $\dot{V}O_{2\ max}$ value (58.2 ± 4.8 ml/kg · min) was approximately 20% below that of their male counterparts and in close agreement with the values for ♀ distance runners recently reported by Wilmore and Brown (1974) – 59.1 ml/kg · min – and Daniels et al. (1977) – 59.6 ml/kg · min. The highest $\dot{V}O_{2\ max}$ value observed on subject RC (aged 25 years, 42.2 km time 3.037 h – Table 1) of 64.4 ml/kg · min in the present study is similar to that reported for elite female cross country skiers (63 ml/kg · min – Saltin and Åstrand, 1967), but is significantly less than the measurement obtained on the world's best female marathon athlete (42.2 km time 2.828 h) of 71.1 ml/kg · min which stands as the highest recorded value of aerobic performance on women to date (Wilmore and Brown, 1974). The directly observed aerobic power outputs in the present study are associated with running performance from 5–84.64 km, but the magnitude of the correlation between the two variables diminishes with increasing distance. From the 5 km times $\dot{V}O_{2\ max}$ may be predicted with a SD of 2 ml/kg · min in the men (Eq. 2) but converting times to speeds and using Eq. 1 reveals that the mean estimated $\dot{V}O_{2\ max}$ is approximately 6% below the observed value (Table 2). However, two important points should be noted, our $\dot{V}O_{2\ max}$ observations were made using a motor driven treadmill, the speed of which was maintained constant and the gradient raised to achieve maximal values. Åstrand and Saltin (1961) has shown that this procedure may give 5% higher values than running on the level. Equally, no account was taken of wind resistance in our experiments. Recently, Pugh (1970) has shown that the O_2 cost of running outdoors against a wind increases as the square function of its velocity. On a calm day the $\dot{V}O_2$ is proportional to the cube of the athletes running speed. On the treadmill, of course, the wind resistance is effectively zero. Thus, over the range of speeds recorded for

the 5 km on the track and using a mean projected area for our athletes estimated from the formula given by Pugh (1970) wind resistance could account for ~ 5% of the $\dot{V}O_2$ of running. Either one or both of these factors could account for the lower estimated $\dot{V}O_{2\,max}$ values from 5 km time given in Table 2, though it should be noted that recently McMiken and Daniel (1976) have failed to demonstrate a difference in $\dot{V}O_2$ between track and level treadmill running. It may be that small differences in efficiencies between performance on the track and treadmill compensate for (or possibly obscure) the effects of wind resistance in the two situations. It is certainly of interest that Pugh's track data agrees precisely with our laboratory observations which were obtained on a traditional (modified Collins) 'rollerbed' treadmill and we are currently investigating this problem further. In the meantime we hold the view that the 5 km time provides a useful guide to the endurance athletes $\dot{V}O_{2\,max}$ and provides a worthwhile physiological tool for field studies.

At the longer distances, as stated, the correlation between $\dot{V}O_{2\,max}$ and running performance diminishes, but again in contrast to Costill (1972), who found the two variables to be unrelated ($r = +0.08$), it does remain significant ($p < 0.001$) at both 42.2 km and 84.64 km (Fig. 3). In the male athletes the correlations between $\dot{V}O_{2\,max}$ and 42.2 km and 84.64 km times were −0.82 and −0.72 respectively. In the females at 42.2 km the association was less strong ($r = -0.43$) but nevertheless remained significant ($p < 0.001$). The residual variance in performance in both sexes as distance increased could be accounted for in terms of relative sustainable work output. In the male athletes $\%\dot{V}O_{2\,max}$ was responsible for 36% and 80% of the total variance of 42.2 km and 84.64 km times, respectively. In the women the respective figure for 42.2 km was 25%. Indeed, multiple regression analysis revealed that using the following formulas:

$$42.2 \text{ km time (h)} = 7.445 - 0.0338\ \dot{V}O_{2\,max}\ (\text{ml/kg} \cdot \text{min}) - 0.0303\ \%\dot{V}O_{2\,max}$$
$(r = 0.993)$,

$$84.64 \text{ km time (h)} = 16.998 - 0.0735\ \dot{V}O_{2\,max}\ (\text{ml/kg} \cdot \text{min}) - 0.0844\ \%\dot{V}O_{2\,max}$$
$(r = 0.996)$.

Of the variance of performance times 97–98% could be accounted for in the two events. This supports the view that other factors such as running technique, clothing, weight of footwear (Costill, 1972) etc., in a temperate climate and at least for the groups of athletes observed in this study, play a relatively minor role in determining overall performance in marathon and ultralong-distance running.

The relationship between $\%\dot{V}O_{2\,max}$ and performance time in long-distance running found in the present study precludes specific statements regarding the relative work load which can be sustained for a given distance by marathon and ultramarathon athletes and may help to explain the contradictory results which have been reported in the literature. For example, Costill and Fox (1969) and Dill (1965) have suggested that the marathon (42.2 km) is run at 75–77% $\dot{V}O_{2\,max}$, longer distances at 60% $\dot{V}O_{2\,max}$ and yet Costill and Winrow (1970) report that two middle-aged runners could sustain 80–85% $\dot{V}O_{2\,max}$ for 2.5 to 5.5 h. Indeed, their subject, McDough, averaged 83.5% $\dot{V}O_{2\,max}$ for 60.4 km and 78% $\dot{V}O_{2\,max}$ for 84.5 km. Costill et al. (1971) later reported that Clayton (still the holder of the world's best

recorded time for the marathon of 2.143 h) was capable of working at 86% $\dot{V}O_{2\ max}$ for prolonged periods. Our data could be used to support all these figures depending on the population of athletics sampled. The present male athletes were capable of maintaining 81–92% and 60–82% of their 5 km running speed for 42.2 and 84.64 km, respectively (Table 2). In terms of O_2 cost this represents 76–87% and 53–76% of their uphill $\dot{V}O_{2\ max}$ values observed in the laboratory. The top marathon runners in the world are probably capable of sustaining even greater relative work outputs during a 42.2 km race (Table 4). Using 5 km time to predict $\dot{V}O_{2\ max}$ (Eq. 2) their $\%\dot{V}O_{2\ max}$ values range from 85–92%. In fact the small differences in relative sustainable work load would appear to be decisive in these athletes, all of whom have a highly developed (and similar) aerobic capacity (Table 4 and Fig. 3). These differences in sustainable $\dot{V}O_{2\ max}$ between the different groups of athletes are undoubtedly in part due to training, but they may also reflect experience. For instance, it will be seen from Table 3 that the women operate at lower $\%\dot{V}O_{2\ max}$ (mean 79% range 68–87%) in the marathon than the men and, indeed, the physiological performance of the female athletes at 42.2 km is closely similar to the men in the longer (London to Brighton) ultramarathon event (Tables 2 and 3 and Fig. 3). In both cases the performance may be explained partly in terms of experience and the prolonged training required to achieve and sustain an aerobic power output close to maximum. It is noteworthy that the two older (and more experienced) female runners (PD and BN, Table 1) were able to sustain approximately 87% of their $\dot{V}O_{2\ max}$ during the marathon (values comparable with the elite male athletes) and thereby compensate for the effects of advancing age on $\dot{V}O_{2\ max}$ (Robinson, 1938; Åstrand, 1960), whereas subject JD, a newcomer to marathon running could only achieve 68% $\dot{V}O_{2\ max}$ (Table 1). Similarly the elite male ultra long distance athlete (CW) achieved approximately 76% $\dot{V}O_{2\ max}$ compared with the 53% $\dot{V}O_{2\ max}$ in KW who was competing in the London to Brighton race for the first time. The marathon is a comparatively new sport for females and in general the male athletes tend to progress to ultramarathon running from the comparatively shorter (16–42 km) events as their athletic career advances. The intersubject differences in sustainable $\%\dot{V}PO_{2\ max}$ can in part be removed by considering time of performance rather than distance (Fig. 4). The relationship between $\%\dot{V}O_{2\ max}$ and time is described by a quadratic equation of the form:

$$\%\dot{V}O_{2\ max} = 91.24 - 3.79 \text{ time (h)} + 0.08 \text{ time}^2 \text{ (h)}.$$

The curve suggests that elite ultralong-distance runners can sustain 87.5 $\%\dot{V}O_{2\ max}$ for 1 h 66.0% for 8 h and 46.4% over a full day and night (24 h) period. These figures should be compared with the data of Costill and Fox (1969) and Åstrand (1960) for trained and untrained subjects, respectively, working for periods up to 8 h and with the statistical predictions of Khosla (1974). The latter author on the basis of early figures concluded that three times the traditional marathon distance could be run non-stop at a speed not exceeding 3 m/s 10.8 km (h) in a time of 11.72 h by some future champion. Five athletes in the present study, DF (11.64 km/h), TR (11.83 km/h), MWT (12.66 km/h), TOR (13.36 km/h), and CW (13.82 km/h) have achieved average speeds well in excess of Khosla's prediction over greater periods of time and distance. Indeed, TR averaged 10.48 km/h (which corresponded to an

Table 4. *Running and estimated aerobic performance of the elite male British marathon athletes. Estimated maximal aerobic power output ($\dot{V}O_{2\,max}$ — from Eq. 1 in text). 5 km time, speed, estimated oxygen intake ($\dot{V}O_2$ from Eq. 2 in text) and relative work load ($\%\dot{V}O_{2\,max}$)*

Athlete	Estimated $\dot{V}O_{2\,max}$ (ml/kg · min)	5 km				42.2 km				
		Time (min)	Speed (km/h)	$\dot{V}O_2$ (ml/kg · min)	$\%\dot{V}O_{2\,max}$	Time (h)	Speed (km/h)	%[a]	$\dot{V}O_2$ (ml/kg · min)	$\%\dot{V}O_{2\,max}$
Thompson	78.8	14.09	21.29	76.7	97.3	2.153	19.60	92.1	70.0	88.8
Hill	79.3	13.94	21.52	77.6	97.9	2.158	19.56	90.9	69.8	88.0
Adcocks	78.2	14.25	21.06	75.8	96.9	2.180	19.36	91.9	69.0	88.2
Alder	78.2	14.24	21.36	77.0	98.5	2.201	19.17	89.8	68.3	87.3
Faircloth	78.2	14.26	21.04	75.7	96.8	2.205	19.14	91.0	68.2	87.2
Stewart	80.4	13.64	21.99	79.5	98.9	2.220	19.00	86.4	67.6	84.1
Wright	80.4	13.65	21.97	79.4	98.7	2.224	18.97	86.4	67.5	84.0
Heatley	79.6	13.85	21.66	78.2	98.2	2.232	18.91	87.3	67.2	84.4
MacGregor	78.5	14.15	21.20	76.3	97.2	2.238	18.86	88.9	67.1	85.5
Plain	80.4	13.64	21.99	79.5	98.8	2.249	18.76	85.3	66.7	83.0
Watson	77.9	14.33	20.93	75.3	96.7	2.252	18.74	89.5	66.6	85.5
Kirkham	77.5	14.45	20.76	74.6	96.3	2.255	18.71	90.1	66.5	85.8
Angus	79.3	13.93	21.53	77.6	97.8	2.265	18.63	86.5	66.1	83.4
Mean	79.0	14.03	21.41	75.0	97.7	2.218	19.03	88.9	67.7	85.8
± SD	±1.0	±0.27	±0.41	±1.0	±0.9	±0.037	±2.32	±2.3	±1.3	±2.0

[a] 42.2 km speed expressed as % of 5 km value

estimated 47% of his $\dot{V}O_{2\ max}$ – Davies and Thompson, 1979) when covering non-stop a distance of 251.5 km over a period of 24 h.

Finally, Pugh et al. (1967) have suggested that a 2 h 10 min run requires a $\dot{V}O_2$ of 61 ml/kg · min. Our data would suggest the figure is nearer 70 ml/kg · min and that modern marathon runners require a $\dot{V}O_{2\ max}$ approaching 80 ml/kg · min to excel at their chosen sport. It is of some interest and surprise that Costill et al. (1971) report that Clayton's $\dot{V}O_{2\ max}$ is 69.7 ml/kg · min which from our data would require him to operate at this value to achieve his world best time at 42.2 km. Costill and his co-workers suggest that Clayton's efficient running style compensated for his relatively "low" $\dot{V}O_{2\ max}$. We cannot comment on this finding directly except to say that though we did find intersubject variation in efficiency (of the order of 5%) this factor was not reflected in their performance time. We have found that the ability to succeed at marathon and ultralong-distance running is almost wholly dependent on a well developed aerobic power output and the capacity to utilise and sustain a given relative work output during competition. This probably explains why individual performances for a specific event are so hazardous to predict in the marathon. For a group of elite athletes, all of whom have a well (and similarly) developed $\dot{V}O_{2\ max}$, the differences in performance will rest on the ability to utilise and sustain their aerobic capacity within very narrow limits (Table 4). This may depend on the day of the race on psychological as well as physiological factors.

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Thermoregulation During Exercise in Relation to Sex and Age

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Summary. The thermoregulatory responses to 1 h exercise of 14 male (age range 18–65 year) and 7 female (age range 18–46 year) athletes and 4 (3♂ and 1 ♀) non-athletic subjects have been investigated in a moderate environment (T_{db} = 21° C, T_{wb} = 15° C and rh < 50%) and analysed in relation to age, sex, and maximum aerobic power output ($\dot{V}O_2$ max).

The maximal sweat loss ($\dot{M}_{sw\ max}$) under the given conditions was closely related (r = + 0.90) to $\dot{V}O_{2\ max}$ and for a given relative work load ($\%\dot{V}O_{2\ max}$), rectal (T_{re}) and mean skin ($\bar{T}_{sk}$) temperatures was the same in all subjects.

Sweat loss ($\dot{M}_{sw}$) was linearly related to total heat production (H) and to peripheral tissue heat conductance (K) and if expressed in relative terms ($\%\dot{M}_{sw\ max}$) was linearly related to T_{re}. For a given T_{re} relative sweat rate was identical in the groups studied. From these results it would seem that during exercise T_{re} rises to meet the requirements of heat dissipation by establishing a thermal gradient from core to skin and stimulating sweating in proportion to maximal capacity of the system. Thus provided the thermal responses to work were standardised using the appropriate physiological variables, there was no evidence to be found for differences in thermoregulatory function which could be ascribed to sex or age.

Key words: Work – Rectal temperature – Skin temperature – Evaporative sweat loss – Age – Sex differences – Maximum aerobic power output – Sweating capacity

There have been several thermoregulatory studies of women at rest before and after acclimatisation, during controlled hyperthermia, and response to standard heat exposure test (see for general review, Kerslake, 1972). The thermal response of women to exercise have been less well documented but data are available from laboratory (Brouha et al., 1960) and field (Dill et al., 1973) investigations. The consensus view from these studies is that women have a higher core temperature for a given heat load and a less efficient sweating mechanism than men. These sex differences are

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said to be due to a higher set point (Bittel and Henane, 1975) for sweat onset and inadequacy of evaporative sweat loss in the transient state before thermal balance is reached which gives rise to increased body heat content and temperature in women particularly when exposed to hot conditions (Wyndham et al., 1965). Interestingly, a similar picture of thermoregulation is described when age is considered. Older men and women are reported to have a poorly developed and insensitive sweat mechanism compared with young people and a low heat tolerance (Ellis et al., 1976). However, despite these scientifically well-supported differences in thermal responses to rest, work, and heat in relation to age and sex, little attention has been directed to the question as to whether they are due to differences of thermoregulatory function per se or are in fact a reflection of the habitual physiological status of the individual. It is well recognised, for example, that the degree of heat acclimatisation (Wyndham et al., 1965) and working capacity (Drinkwater et al., 1976; Piwonka and Robinson, 1976) will affect the thermal responses to a given heat load in both men and women, and under normal conditions both these factors will be affected by the habitual activity of the individual. Since we know that maximum aerobic power output ($\dot{V}O_{2\ max}$) and physical activity decrease with age (Robinson, 1938) and women have a lower $\dot{V}O_{2\ max}$ than men (Åstrand, 1960), it is entirely conceivable that when account is taken of the physical status of the individual, the age and sex differences in thermal function will cease to exist.

The purpose of the present investigation was to examine the association of $\dot{V}O_{2\ max}$ and sweating capacity with the thermal responses to different metabolic heat loads in a moderate environment in subjects who differed in age and sex, but had similar backgrounds of daily habitual physical activity.

Methods

Nine young male, five older male and seven young female athletes were studied during work for 1 h on a motor-driven treadmill, Their physical characteristics are given in Table 1. For interest, data were also collected on 3 young men, 1 middle-aged man, and 1 young girl. These subjects were all active (particularly the men) but not in training, and their results have been included in the analysis at the appropriate level. All the subjects were studied under standardised environmental conditions; T_{db} = 21° C, T_{wb} = 15° C, rh = < 50%, with wind speeds (2.5–5.0 m/s) appropriate for the evaporation of sweat from the skin. A total of 63 experiments were performed. Rectal temperature (T_{re}) was measured at rest and continuously during exercise by means of a thermistor probe inserted 8 cm above the anal

Table 1. Physical characteristics of the subjects. Age, weight (wt), height (ht), and maximal aerobic power output ($\dot{V}O_{2\ max}$). Mean (± SD) data

Subjects		Age (year)	Wt (kg)	Ht (cm)	$\dot{V}O_{2\ max}$ (l/min)
1. ♂ Athletes	($n = 9$)	31.8 ± 4.2	66.4 ± 7.3	175.8 ± 7.5	4.79 ± 0.38
2. ♀ Athletes	($n = 7$)	29.6 ± 8.0	52.3 ± 2.6	162.5 ± 2.2	3.07 ± 0.24
3. ♂ Older athletes	($n = 5$)	60.2 ± 10.4	55.0 ± 10.5	163.1 ± 13.1	3.32 ± 0.48
4. ♂ Non-athletes	($n = 4$)	28.8 ± 10.4	68.0 ± 4.9	181.4 ± 7.3	3.95 ± 0.13

sphincter and skin temperature was measured at 15 min intervals during exercise using a thermocouple bead probe mounted on a plastic applicator. Readings at 13 sites: hand, upper and lower arm, forehead, pectoral, sternum, abdomen, scapula, lumbar, anterior and posterior thigh, and anterior and posterior calf, were recorded and weighted after the method of Hardy and Duboid (1938). The average weighted value of the 13 sites was taken as the mean skin temperature ($\bar{T}_{sk}$). Oxygen intake ($\dot{V}O_2$) was measured using an open circuit technique during the 20–25th min and 50–55th min of exercise. The O_2 and CO_2 content of the expired air was determined using physical (paramagnetic and infra red) analysers which were regularly calibrated with gases of known concentrations from prior Lloyd-haldane chemical analysis. The subjects were weighed clothed before, at the 30 min during, and immediately after exercise on a man-balance accurate to within ± 5 g. The total nude weight loss resulting from the exercise period was also recorded. From these weight loss changes the total sweat production ($\dot{M}_{sw}$) was determined and from the body temperature and metabolic data the total (H) and metabolic (M) heat productions, heat storage (S), and body conductance (K) were calculated using standard equations (see Nielsen, 1969). In 14 subjects (4 ♀ and 4 ♂ athletes, 3 non-athletic and 3 older men) the maximal sweat production ($\dot{M}_{sw\,max}$) for exercise under the stated environmental conditions was determined after the method of Davies (1979), which uses the curvilinear relationship between $\dot{M}_{sw}$ and T_{re}. Essentially during work of different (and increasing) severity but constant (1 h) duration, in a given environment, $\dot{M}_{sw}$ approaches an asymptotic value at $T_{re} > 39.3°$ C (Wyndham, 1965). Thus, the highest value of $\dot{M}_{sw}$ recorded as a result of exercise at $T_{re} > 39.3°$ C was taken as the $\dot{M}_{sw\,max}$ for these subjects. Maximal aerobic power output ($\dot{V}O_{2\,max}$) was measured on all subjects during separate experiments using criteria and methods previously described (Davies, 1968).

Results

The basic data for the 4 groups of subjects are summarised in Table 2. For a given M, rectal temperature (T_{re}) was higher in the female than male athletes. In the older men, T_{re}/M relationship was similar to that found for the ♀ athletes. However, these age and sex differences in T_{re} were resolved if relative work load was considered (Table 2). The rise in T_{re} during exercise at given $\%\dot{V}O_{2\,max}$ was similar in the young (♂ and ♀) and older (♂) subjects (Fig. 1) and the relationship of T_{re} at the 60th min of exercise to $\%\dot{V}O_{2\,max}$ was almost identical to that found previously (Davies et al., 1976) in normal trained and untrained subjects. The presented data could be represented by the equation of the form:

$$T_{re}\ (°C) = 37.39\ (°C) - 0.0018\ \%\dot{V}O_{2\,max} + 0.0003\ \%\dot{V}O^2_{2\,max}. \qquad (I)$$

$\bar{T}_{sk}$ averaged 27.76 ± 1.02° C in the ♀ athletes, 28.39 ± 1.59° C in the ♀ athletes, 27.99 ± 1.19° C in the older subjects and 28.24 ± 1.05° C in non athletic males. There was no significant difference between the groups. Tissue heat conductance (K) was significantly ($P < 0.01$) higher in the male athletes than in the other groups studied due to their higher level of absolute heat production (Table 2). There were no consistent differences in S between the groups studied. $\dot{M}_{sw}$ was closely related to H in all subjects:

$$\dot{M}_{sw}\ (w/m^2) = -\ 30.68 \pm 0.345\ H\ (w);\ r = 0.89 \qquad (II)$$

but for given T_{re}, $\dot{M}_{sw}$ was lower in the females and elderly athletic subjects compared with the male athletes. However, these differences were associated with the $\dot{V}O_{2\,max}$ and $\dot{M}_{sw\,max}$ of the subjects. Under the environmental conditions of the

Table 2. Oxygen intake ($\dot{V}O_2$), relative work load ($\%\dot{V}O_{2\,max}$), metabolic (M), and total heat (H) productions, rectal temperature (T_{re}), mean skin temperature ($\bar{T}_{sk}$), sweat loss ($\dot{M}_{sw}$) and peripheral tissue heat conductance (K) in older and young male athletes, female athletes and non-athletes (Mean ± SD data)

Subjects	$\dot{V}O_2$ (l/min)	$\dot{V}O_{2\,max}$ (%)	M (W)	H (W)	T_{re} (°C)	$\bar{T}_{sk}$ (°C)	$\dot{M}_{sw}$ (w/m²)	K (w/m² · °C)
1. Athletes (♂) ($n = 9$)	3.64[a] ± 0.47	76 ± 6	1242[a] ± 160	1224[a] ± 163	39.13 ± 0.50	27.76 ± 1.02	370[b] ± 54	51.4[b] ± 7.2
2. Athletes (♀) ($n = 7$)	2.39 ± 0.09	78 ± 6	814 ± 32	812 ± 31	39.14 ± 0.47	28.39 ± 1.59	279 ± 29	41.8 ± 9.1
3. Athletes (older ♂) ($n = 5$)	2.48 ± 0.44	76 ± 5	858 ± 147	785 ± 117	39.10 ± 0.23	27.99 ± 1.19	352 ± 157	39.2 ± 7.4
4. Non-athletes (♂) ($n = 4$)	3.01 ± 0.17	76 ± 4	1027 ± 58	905 ± 69	39.18 ± 0.28	28.24 ± 1.05	269 ± 17	39.6 ± 5.4

Significance: Group comparisons, 1 with 2, 3, and 4; [a] $P < 0.001$; [b] $P < 0.01$

present experiments $\dot{M}_{sw\,max}$ (in 14 subjects – see Methods) was found to be a linear function of $\dot{V}O_{2\,max}$:

$$\dot{M}_{sw\,max}\ (w/m^2) = -\ 62.4 \pm 0.50\ \dot{V}O_{2\,max}\ (w/m^2);\ r = +\ 0.90 \qquad \text{(III)}$$

thus, if the relative sweat rate $\left(\%\dot{M}_{sw\,max} = \dfrac{\dot{M}_{sw} \times 100}{\dot{M}_{sw\,max}}\right)$ is plotted as a function of either T_{re} or $\%\dot{V}O_{2\,max}$ the differences between the groups disappear (Fig. 2).

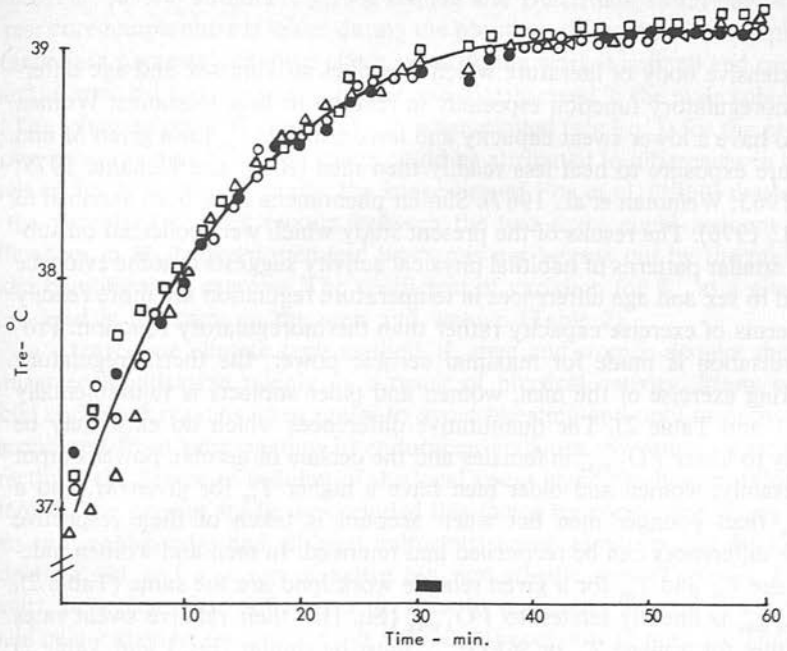

Fig. 1. The time course of rectal temperature (T_{re}) changes during exercise at a given relative ($\%\dot{V}O_{2\,max}$) work load for a representative subject from each of the 4 groups studied. A female (▲), young (●) and older male (□) athlete, and an active (non-athletic) male (○). Exercise was interrupted briefly (■) at the 30th min in order to weight the subjects (see Methods)

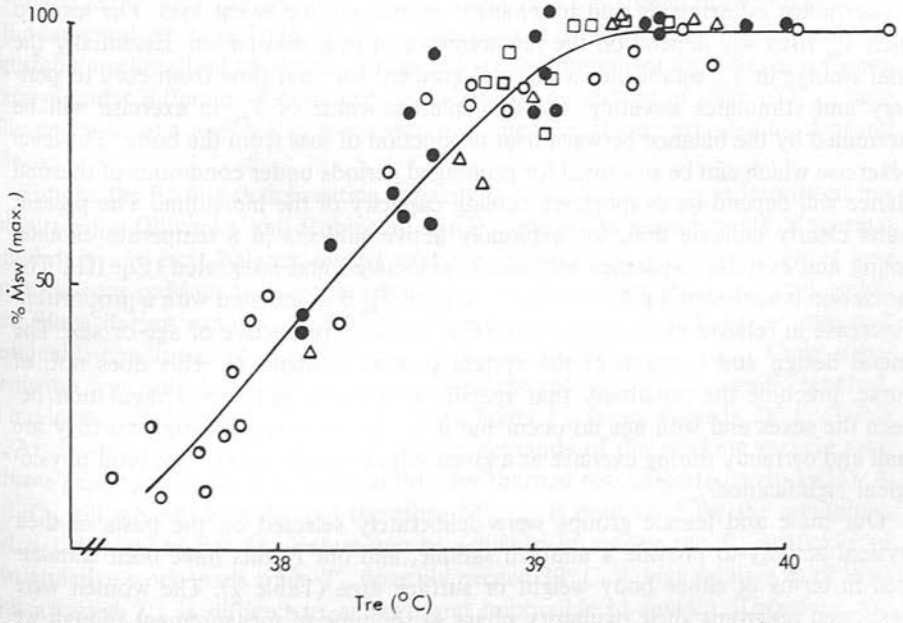

Fig. 2. Relative sweat rate ($\%\dot{M}_{sw\,max}$) in relation to T_{re}. The data represent 63 1-h experiments (2 points have been omitted due to overlap) performed on 7 female (▲), 9 male (●), and 5 older male (□) athletes; 4 male (○) and 1 female (△) non-athletes

Discussion

There is an extensive body of literature which describes striking sex and age differences in thermoregulatory function especially in relation to heat tolerance. Women are reported to have a lower sweat capacity and have a higher T_{re} for a given M and therefore endure exposure to heat less readily then men (Bittel and Henane, 1975; Hertig et al., 1963; Weinman et al., 1967). Similar phenomena have been ascribed to age (Ellis et al., 1976). The results of the present study which were collected on subjects who had similar patterns of habitual physical activity suggests that the evidence so far ascribed to sex and age differences in temperature regulation are more readily explained in terms of exercise capacity rather than thermoregulatory function. Provided standardisation is made for maximal aerobic power, the thermoregulatory behaviour during exercise of the men, women and older subjects is fundamentally similar (Fig. 1 and Table 2). The quantitative differences which do exist may be related directly to lower $\dot{V}O_{2\ max}$ in females and the decline of aerobic power output with age. Certainly, women and older men have a higher T_{re} for given M, and a lower $\dot{M}_{sw\ max}$ than younger men but when account is taken of their respective $\dot{V}O_{2\ max}$, these differences can be reconciled and removed. In men and women independently of age T_{re} and $\bar{T}_{sk}$ for a given relative work load are the same (Table 2), and since $\dot{M}_{sw\ max}$ is linearly related to $\dot{V}O_{2\ max}$ (Eq. III), their relative sweat rates ($\%\dot{M}_{sw\ max}$) either for a given T_{re} or $\%\dot{V}O_{2\ max}$ must be similar (Eq. I and Table 2) thus, these responses must be regarded, not in terms of age or sex, but as reflecting normal physiological thermoregulatory function.

The human thermoregulatory system is designed to maintain core temperature independently of $\bar{T}_{sk}$ and environmental conditions. During exercise this is achieved by vasomotor adjustments and by changes in evaporative sweat loss. The level to which T_{re} rises will depend on the requirements of heat dissipation. Essentially the initial change in T_{re} establishes a thermal gradient for heat flow from core to periphery and stimulates sweating, the final plateau value of T_{re} in exercise will be determined by the balance between heat production of loss from the body. The level of exercise which can be sustained for prolonged periods under conditions of thermal balance will depend on evaporative cooling capacity of the individual. The present results clearly indicate that, for habitually active subjects in a temperate climate, cooling and exercise capacities are closely associated and integrated (Eq. III). The association is such that a given rise in T_{re} at a set $\bar{T}_{sk}$ is associated with a proportional increase in relative sweat production (Fig. 2), i.e., irrespective of age or sex. The general design and function of the system in man is identical. This does not, of course, preclude the possibility that specific differences in thermal regulation between the sexes and with age do occur but if so, the present data suggests they are small and certainly during exercise at a given relative work level of doubtful physiological significance.

Our male and female groups were deliberately selected on the basis of their physical activity to provide a uniform sample, and our results have been standardised in terms of either body weight or surface area (Table 2). The women were questioned regarding their ovulatory phase at the time of measurement though we did not investigate this problem. No girl was menstruating during the period of their laboratory visits and 4 of the 7 girls were investigated in the post-ovulatory phase of

their cycle. Several authors (e.g., see Haslag and Hertzman, 1965) have shown that at rest core temperature is lower during the pre- than the post-ovulatory phases. The present data suggests that if the effect exists during work it is small and certainly less than the inter-subject variability of exercise T_{re} observed in the male subjects (Table 2). The relationship of T_{re} to $\%\dot{V}O_{2\,max}$ when plotted (see Eq. I) for the present data shows no systematic variation which could be attributed to differences in circulating levels of sex hormones. Equally, the suggestion of Fox et al. (1969) that differences in the vascularity of the rectum between the two sexes could account for larger differences in T_{re} between men and women is not bourne out by the present study under conditions of exercise. The coefficient of variation for T_{re} at a given relative work load is the same in the men and women (Table 2).

In a temperate climate such as the UK, men and women acquire their state of natural acclimatisation mainly as a result of physical activity. Many women for social and other reasons often prefer to avoid sweating and until recently have been discouraged from participating in endurance activities. Sweating capacity reflects directly to the degree of training of the local sweat gland mechanism (Collins et al., 1966). In the present study we excluded this factor by comparing young and older men and women who had all been uniformly active. However, we did include one sedentary girl and a group of active but non-athletic men (Table 2). From their results, it is clear that differences do exist in maximum sweating capacity which could be ascribed to sex and age but this would serve only to mislead. The $\dot{M}_{sw\,max}$ is a function of $\dot{V}O_{2\,max}$ (Eq. III) and the thermoregulatory adjustments of the individual are in accord with his capacity not only to dissipate but produce metabolic heat. Naturally, if exposed to heat it is to be expected that the level of tolerance which itself is a function of sweating capacity will be associated with age and sex as described in the literature (Wyndham et al., 1965), but this should not be regarded as a fundamental or causal relationship. The present analysis suggests for subjects 'naturally' acclimatised by their habitual activity thermoregulatory behaviour during exercise under different climatic conditions will be a function of their $\dot{V}O_{2\,max}$ and will vary with age and sex in a manner described for these variables by Åstrand (1960).

Finally, the finding that sweating capacity is related to $\dot{V}O_{2\,max}$ is important but not surprising (Piwonka and Robinson, 1967). Indeed, the requirements of thermal equilibrium and heat balance during exercise demand that evaporative sweat loss and total heat production must be related linearly for a given environmental condition. Since during running on the level for 1 h, $\tilde{H} = M_0 = \dot{V}O_2$ under maximal sustainable conditions H_{max} and $\dot{M}_{sw\,max}$ must be a function of $\dot{V}_{O2\,max}$. These statements are true only for environments in which thermal equilibrium can be reached. In moderate environments and at high work loads $\bar{T}_{sk}$ rarely exceeds 28° C (Table 2). At these levels of exercise the metabolic demands of the working muscle may achieve priority over peripheral blood flow for thermal regulation (Rowell, 1974). A low $\bar{T}_{sk}$ will inhibit sweating and therefore $\dot{M}_{sw\,max}$ is only valid for the conditions stated. Certainly higher $\dot{M}_{sw}$ values can be achieved by raising the $\bar{T}_{sk}$ artifically at lower relative work loads when T_{re} does not exceed 39° C. A high relative work load with a raised $\bar{T}_{sk}$ is difficult to achieve and impossible to sustain. Therefore, the relationship of $\dot{M}_{sw\,max}$ to $\dot{V}O_{2\,max}$ (Eq. III) found in this study must only be applied to the conditions under which it was measured (cf. Fox et al., 1969). If this is accepted

then the new concept introduced by the present study is that of *relative sweat rate* ($\%\dot{M}_{sw\ max}$). This gives further insight into thermoregulatory physiology during exercise in moderate environmental conditions. A set rise in core temperature promotes a given proportional increase in $\%\dot{M}_{sw\ max}$ (Fig. 2). This may in part explain the large intersubject variability of T_{re} when related to M (Davies et al., 1976) and the close association of T_{re} with $\%\dot{V}O_{2\ max}$ (Saltin and Hermansen, 1966). The rise in core temperature promotes a proportionate increase in sweat rate to meet the requirements of given relative (metabolic) heat production. On this basis one might expect to be able to predict an individual's thermal and physiological responses to work in different environments if the $\dot{V}O_{2\ max}$ is known. However, some caution is necessary. $\dot{M}_{sw\ max}$ is associated but not causally related to $\dot{V}O_{2\ max}$ and undoubtedly the power output of the sweating and aerobic mechanisms can be trained or changed independently. Further role of physical activity in maintaining vasomotor tone cannot be ignored. This factor is probably critically important when age is considered. Ageing is often accompanied by a decrease in physical activity which may well fall below the level necessary for maintenance of active vasomotor adjustments and sweating which are essential to normal thermoregulation in a variable climate. The role of systematic programmes of physical activity in ameliorating the decline of vasomotor and sweating function which is known to occur with age, has not been investigated. The present study would suggest that these changes are not inevitable. Regular physical activity may help to offset the risks the elderly suffer from both hyper- and hypothermia and enable them to retain their capacity for normal physiological function and independent living.

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Influence of skin temperature on sweating and aerobic performance during severe work

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DAVIES, C. T. M. *Influence of skin temperature on sweating and aerobic performance during severe work.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 47(4): 770–777, 1979.—Two male subjects were measured over a range of work intensities at dry-bulb temperature (T_{db}) = 21°C (relative humidity, rh <50%) and at approximately 65 and 85% $\dot{V}O_{2\,max}$ for 1 h at T_{db} at 5, 10, 15, 21, and 25°C with high convective airflow (2.5–5 m/s). The results showed that mean skin temperative ($\bar{T}_{sk}$) was related to T_{db} and unaffected by rh over the range studied. $\bar{T}_{sk}$ was dependent on the relative work load and was 2.5°C lower at 85% than 65% $\dot{V}O_{2\,max}$ in the cooler environments. During submaximal work the relative sweat rate ($\dot{M}_{sw}$ expressed as $\%\dot{M}_{sw,\,max}$) was a linear function of rectal temperature (T_{re}) and $\bar{T}_{sk}$ for each subject and thus $\%\dot{M}_{sw,\,max}$ could be predicted from these two variables with a standard deviation of ±12%. For a given $\bar{T}_{sk}$, T_{re} appeared to rise to meet the requirement of heat loss by stimulating set $\%\dot{M}_{sw}$ response. However, during severe work (85% $\dot{V}O_{2\,max}$) this mechanism appeared to become saturated, T_{re} (except for a very narrow prescriptive range) was dependent on T_{db}. These results suggest that under moderate environmental conditions the maximal aerobic and evaporative (cooling) power outputs of an individual are closely matched and only during extremely hard work does thermoregulation become passive and effectively physical (rather than physiological) in nature.

evaporative sweat loss; maximum aerobic power; rectal and skin temperatures; exercise; thermal regulation; control of sweating

IT HAS BEEN SUGGESTED that during prolonged severe exercise in moderate environments the heat dissipating mechanisms become saturated probably as a result of cardiovascular overload (3, 20). In a study from this laboratory (3) at work loads beyond 85% maximal aerobic power ($\dot{V}O_{2\,max}$), which could be sustained for 1 h, there was evidence of vasoconstriction; the skin remained cool (mean skin temperature [$\bar{T}_{sk}$] rarely exceeded 28°C), but as work progressed there was a spiraling increase on rectal temperature (T_{re}). Thermal control during the latter stages of this type of exercise (equivalent to marathon running) appeared to be "passive", thus we postulated that it would be extremely sensitive to small changes in climatic conditions. Because the dry-bulb temperature (T_{db}) in our experiments did not exceed 22°C and relative humidity (rh) was <50%, it was thought of interest to extend the observations of temperature regulation during exercise of high intensity and prolonged duration to a wider range of environments. The present investigation is therefore concerned with the influence of T_{db} on T_{re}, $\bar{T}_{sk}$, sweating, and aerobic performance during work at constant speed on a motor-driven treadmill.

METHODS

Two male subjects were studied. *Subject 1* (wt, 65 kg; ht, 173 cm; $\dot{V}O_{2\,max}$, 4.72 l/min) was an international ultralong-distance runner in regular training. *Subject 2* (wt, 62 kg; ht, 178 cm; $\dot{V}O_{2\,max}$, 3.8 l/min) ran regularly for pleasure, but not competitively. Twenty-four exercise experiments were carried out at various relative work loads in a constant environment of T_{db} 21°C and 16 experiments were conducted ~65% $\dot{V}O_{2\,max}$ and ~85% $\dot{V}O_{2\,max}$ at 5, 10, 15, and 25°C. Relative humidity was always less than 50% and the tolerance limits for environmental temperatures in the climatic chamber was of the order of ±1°C. Airflow was maintained to ensure the evaporation of sweat from the skin and ranged from 2.5–5 m/s in the two subjects at the higher environmental temperatures, but was reduced to <2 m/s in the cooler conditions. In some additional experiments on *subject 2*, whose maximal sweat rate was ~two-thirds that of *subject 1*, the skin was sprayed with a thin film of water at a temperature to match the existing $\bar{T}_{sk}$ and at a frequency sufficient to allow full evaporation. The air velocity was maintained at a constant rate of 2.5 m/s. The exact volume of water added to the skin throughout the experiment was monitored; the data for these observations are presented separately in Fig. 8. In all experiments the subjects exercised for 1 h and the following measurements were taken.

Metabolism. The metabolic heat production (M) was calculated from oxygen intake ($\dot{V}O_2$) measured by the open-circuit technique at the 20th and 50th min of exercise using the calorific equivalent of O_2 for a given exchange ratio (R) from standard tables.

Body temperatures. Deep body temperature was measured by a probe inserted 8 cm into the rectum. Skin temperatures were measured using a thermocouple mounted across the open end of a plastic applicator held lightly against the skin. The T_{sk} was measured at 13 sites as previously described (3).

Body weight. Nude and clothed weight was measured immediately prior to exercise on a beam balance accurate to ±5 g. Clothed weight was taken after 30 min of exercise and again immediately on cessation of effort. Nude weight was measured within 5 min of the end of exercise

following the removal of any unevaporated sweat with a towel. Evaporative sweat loss ($\dot{M}_{sw}$) was calculated as the total clothed weight loss after correction for metabolic gas exchange and respiratory water loss and expressed in $W \cdot m^{-2}$.

The heat production (H) was calculated as M minus the vertical work performed (W) on the motor-driven treadmill. For *subject 1* at 21°C the gradient of the treadmill ranged from 0–4% and the speed from 9.7–14.5 km/h. In *subject 2* the respective figures were 0–15% and 6.4–11.2 km/h. The 65% and 85% $\dot{V}O_{2\,max}$ loads in the different environmental conditions corresponded to 0% (14 km/h) and 3% (14.5 km/h) in *subject 1* and 1% and 5% at 11.3 km/h in *subject 2*. The tissue heat conductance (K) was calculated from the formula

$$K = \frac{H_{sk}}{(T_{re} - \bar{T}_{sk}) \times A_D} \quad (W \cdot m^{-2} \cdot °C^{-1}) \qquad (1)$$

A_D, body surface area; H_{sk}, the heat dissipated from the skin, namely H corrected for respiratory heat loss from the lungs and body heat storage (S). The mean skin temperature ($\bar{T}_{sk}$) was computed from the 13 sites using the formula and weighting factors given by Hardy and Dubois (6). Value of T_{re} and $\bar{T}_{sk}$ at the 45th and 60th min of exercise were used in the equation. Heat storage was calculated from the equation: $S = 0.83\ W\ (0.9\ \Delta\ T_{re} + 0.1\ \Delta\ \bar{T}_{sk})$, where 0.83 is the specific heat of the body tissues in kcal and W is body weight in kg and then converted to $W \cdot m^{-2}$. Maximal aerobic power output ($\dot{V}O_{2\,max}$) was determined in separate experiments using the criteria and methods previously described (3).

RESULTS

Figure 1 shows plot of T_{re} against time for work of 85 and 65% $\dot{V}O_{2\,max}$ at 5 and 21°C in *subject 2*. At the lower relative work load, T_{re} reaches a plateau value independently of T_{db}, but at 85% $\dot{V}O_{2\,max}$ this is no longer true. At 21°C, T_{re} rises after 20 min of exercise as a linear function of time to reach a final value approximately 1.4°C higher than observed at 5°C. When plotted against T_{db} (Fig. 2), T_{re} at 85% $\dot{V}O_{2\,max}$ shows an 'S'-like curve suggesting a very narrow prescriptive (cf. Lind, Ref. 9) range over which T_{re} is actively regulated during this severe form of work. At T_{db} <10°C and >20°C, T_{re} is affected by changes in ambient temperature. This effect is not apparent at 65% $\dot{V}O_{2\,max}$ (Fig. 2). The effect of T_{db} is to linearize the relationship of T_{re} to $\%\dot{V}O_{2\,max}$ (Fig. 3).

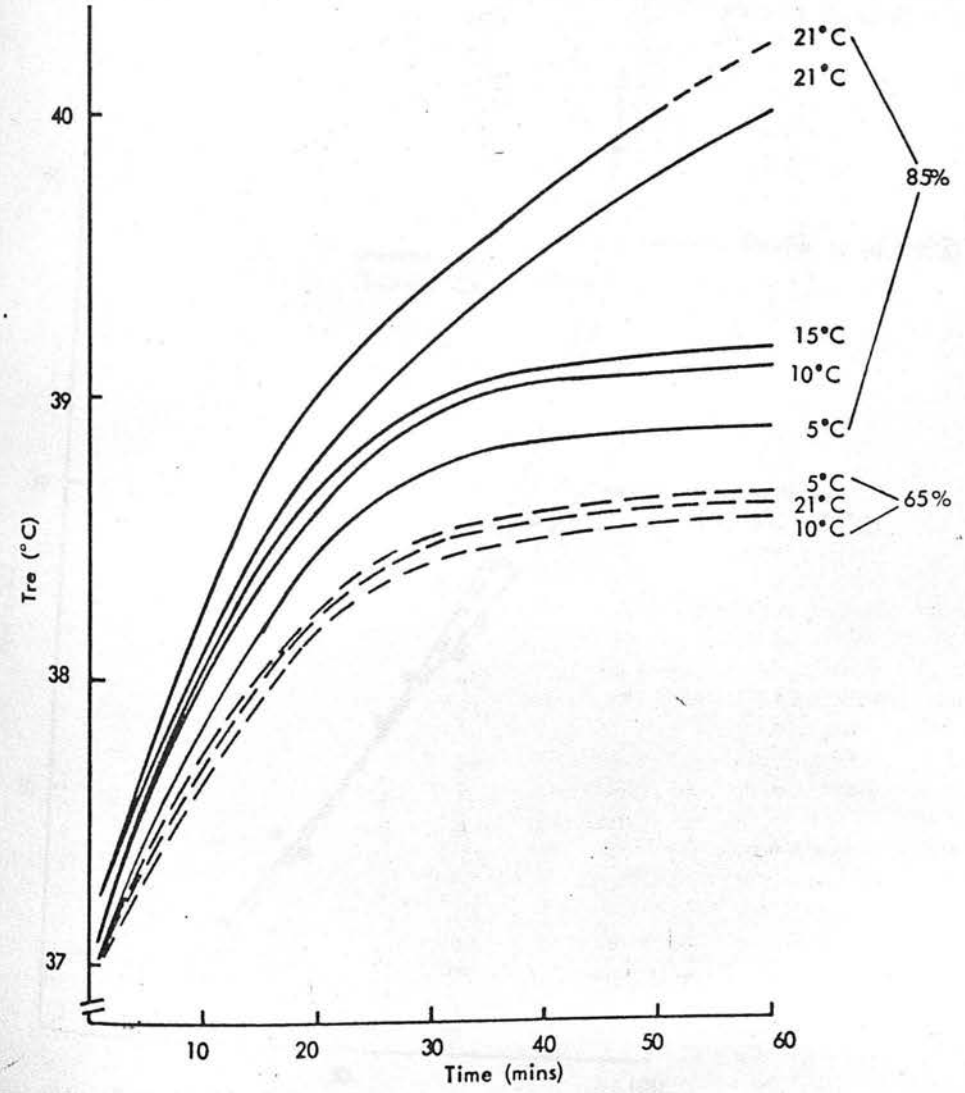

FIG. 1. Relationship of rectal temperature (T_{re}) to time at approximately 85% (*solid line*) and 65% (*dotted lines*) $\dot{V}O_{2\,max}$ for T_{db} of 5, 10, 15 and 21°C. *Subj 2* was unable to exercise at 25°C at 85% $\dot{V}O_{2\,max}$ for 1 h. At 21°C he failed at the 50th min of exercise on 1 occasion, hence dotted portion of the curve.

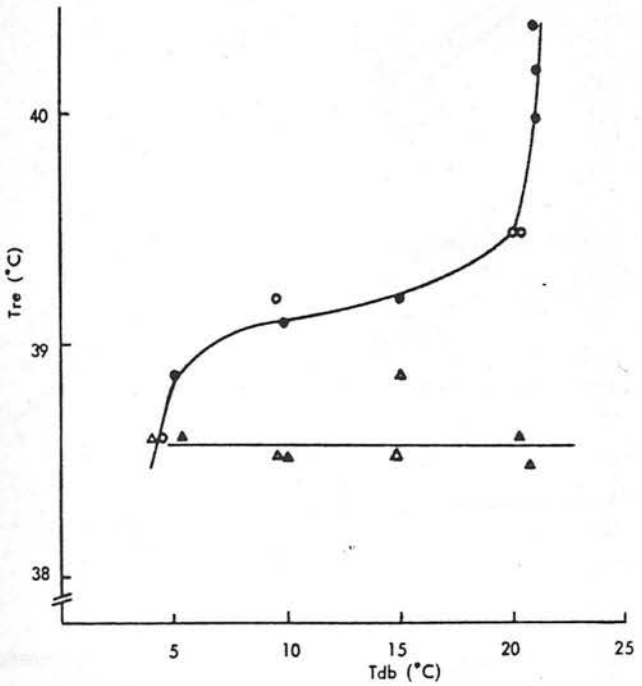

FIG. 2. Rectal temperature (T_{re}) in relation to dry-bulb (T_{db}) temperature at approximately 85% (●, *subj 2*; ○, *subj 1*) and 65% $\dot{V}O_{2\,max}$ (▲, *subj 2*; △, *subj 1*).

Skin temperature. Figure 4 shows the relationship of $\bar{T}_{sk}$ to T_{db} at moderate (65% $\dot{V}O_{2\,max}$) and the most severe (85% $\dot{V}O_{2\,max}$) work loads. In both cases the relationship is linear. The effect of increasing the work output is to increase the slope of the $\bar{T}_{sk}/T_{db}$ relationship so that at 5°C the $\bar{T}_{sk}$ is 2.5°C lower at 85% $\dot{V}O_{2\,max}$ than at 65% $\dot{V}O_{2\,max}$. Humidity, in the presence of high airflow, over the limited range (see METHODS) studied in this investigation, appears to have no effect on the $\bar{T}_{sk}/T_{db}$ relationship. During exercise $\bar{T}_{sk}$ invariably fell at the onset of work then rose slightly, but was usually constant from the 15th–60th min of work. However, there were large regional variations in skin temperature, particularly during work in the cool and cold environments.

Tissue heat conductance. Tissue heat conductance varied from 25.8 to 39.3 $W \cdot m^{-2} \cdot °C^{-1}$ in *subject 2* and 31.8 to 50.4 $W \cdot m^{-2} \cdot °C^{-1}$ in *subject 1* over the range of exercise intensities studied. In both subjects K was associated with $\bar{T}_{sk}$ and metabolism and curvilinearly related to T_{re}. At T_{re} higher than 39.3°C, K tended to plateau with no further significant rise (Fig. 5).

Evaporative sweat loss. Evaporative sweat loss was linearly related to H; thus $\dot{M}_{sw}$ ($W \cdot m^{-2}$) = − 66.11 + 0.38 H (W), r = + 0.64 (*Eq. 1*) and showed the same relationship to T_{re} as K (Fig. 5). Thus the relationship of evapo-

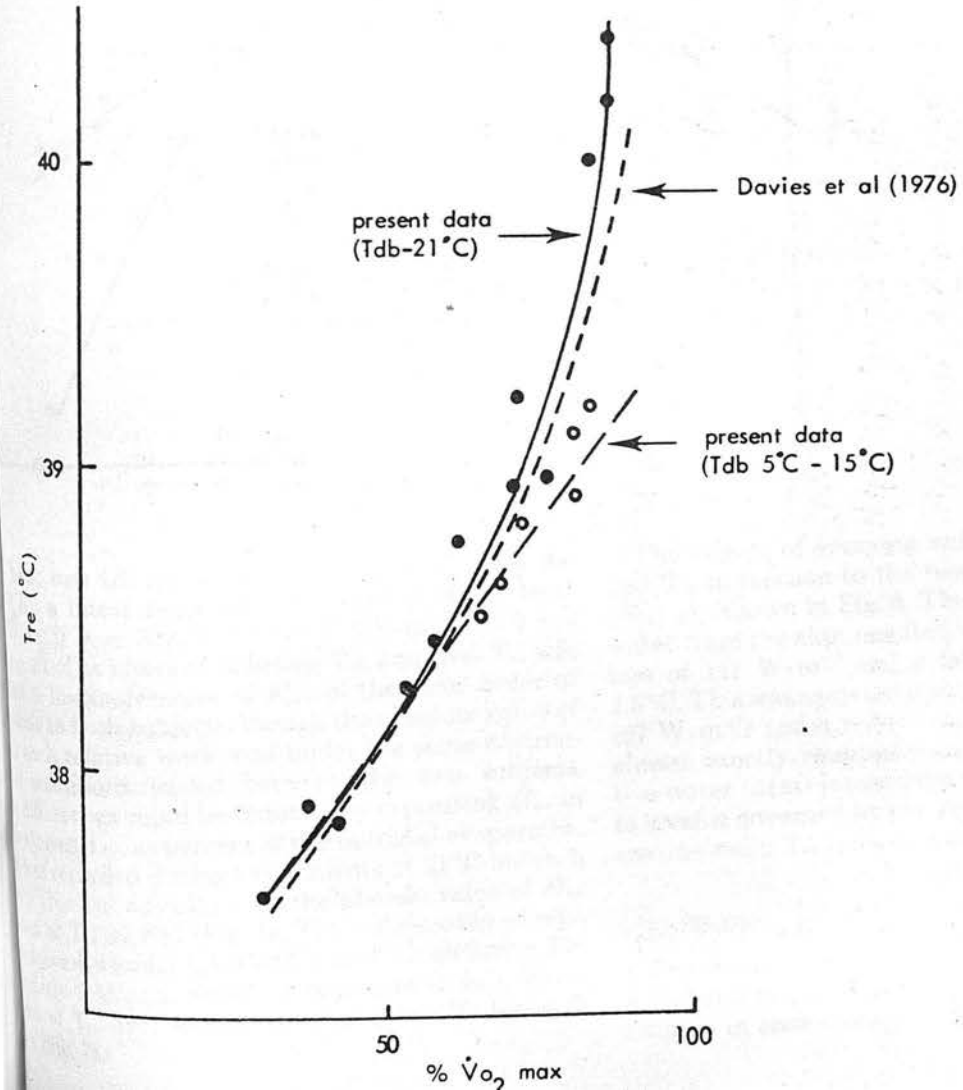

FIG. 3. Rectal temperature (T_{re}) in relation to relative aerobic power output (%$\dot{V}O_{2\,max}$) T_{db} = 21°C (●) and T_{db} = 5–15°C (○) for *subj 2*.

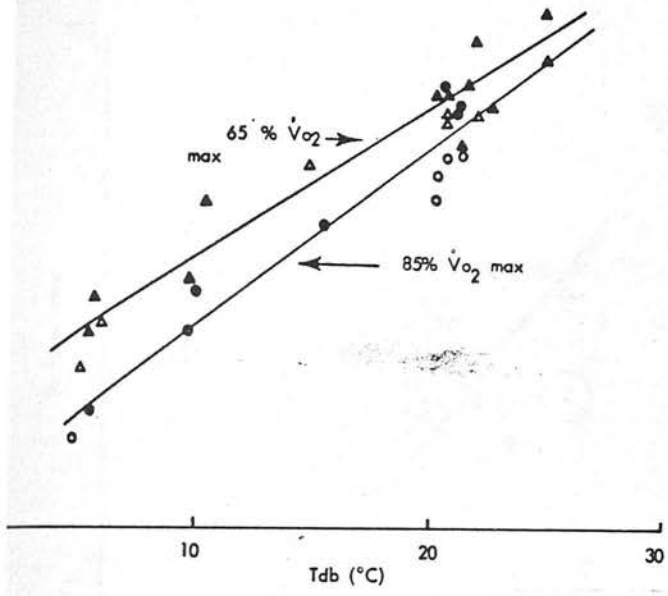

FIG. 4. Mean skin temperature ($\bar{T}_{sk}$) in relation to ambient temperature at 65% $\dot{V}O_{2\,max}$ and 85% $\dot{V}O_{2\,max}$. Symbols same as Fig. 2. Relationship for the 2 relative work loads can be described by the following linear regression equations: $\bar{T}_{sk}$ (°C) = 21.70 + 0.299 T_{db} (°C), r = + 0.96 (65% $\dot{V}O_{2\,max}$) and $\bar{T}_{sk}$ (°C) = 19.60 + 0.367 T_{db} (°C), r = + 0.97 (85% $\dot{V}O_{2\,max}$).

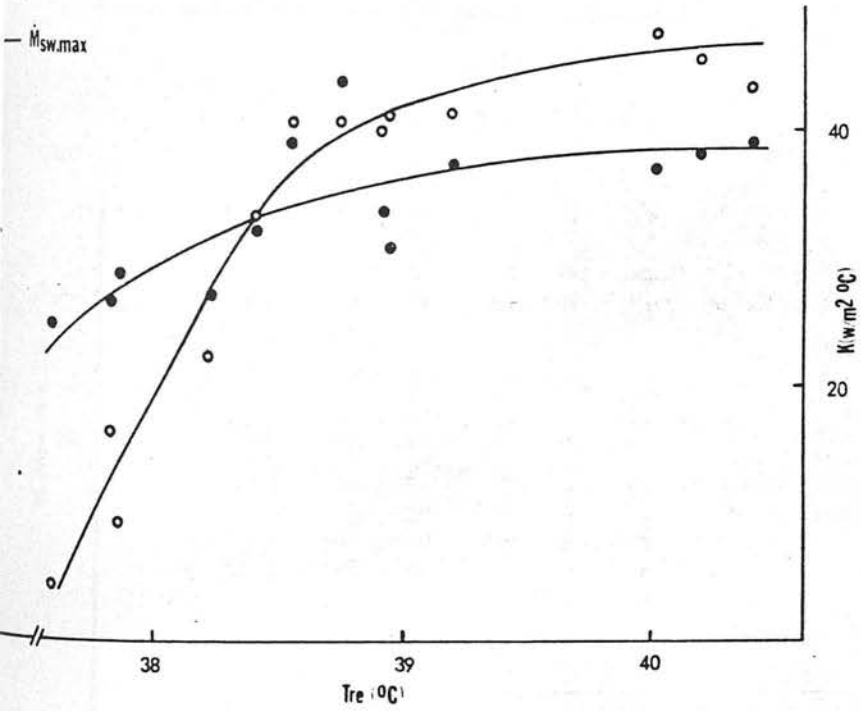

FIG. 5. Evaporative sweat loss (○) and peripheral tissue heat conductance (●) in relation to T_{re}. Data for *subj 2*. *Arrowed line*: predicted $\dot{M}_{sw,max}$ for subject (see text).

heat loss (E) to K was therefore adequately de- l by a linear regression relationship of the form $W \cdot m^{-2}$) = − 209.72 ± 11.36 K ($W \cdot m^{-2} \cdot °C^{-1}$) r = q. 2). The effect of reducing T_{db} and thus $\bar{T}_{sk}$ was ct a linear decrease of $\dot{M}_{sw}$ of the same order of tude in both subjects, though the absolute value of each relative work load under the same environ- l conditions varied between the two subjects. differences could be reduced by expressing $\dot{M}_{sw}$ in e terms, i.e., as percent of the maximal evaporative loss recorded during experiments at 21°C for each t. This was calculated as the plateau value of $\dot{M}_{sw}$ ed at T_{re} 39.3°C (Fig. 5). The relationship of rela- aporative sweat loss ($\%\dot{M}_{sw}$) to $\bar{T}_{sk}$ is shown in Fig. ilarly $\%\dot{M}_{sw,max}$ could be represented as a linear on of T_{re} over the range of 37.0–39.3°C for each t (Fig. 7).

The effects of spraying *subject 2* with water on $\dot{M}_{sw}$ and $\bar{T}_{sk}$ in relation to the heat dissipated from the skin (H_{sk}) are shown in Fig. 8. The evaporation of the film of water from the skin resulted in an additional mean heat loss of 117 $W \cdot m^{-2}$ and a fall in $\bar{T}_{sk}$ of approximately 2.5°C. This was associated with a suppression of sweating (67 $W \cdot m^{-2}$) and a reduction in $C + R$ (45 $W \cdot m^{-2}$) that almost exactly counterbalanced the increased evaporative water (heat) losses from the skin. The $\bar{T}_{sk}$ decreased to a value governed by the overall cooling capacity of the environment; T_{re} remained unchanged.

DISCUSSION

The results of the present study show that at very high work loads in excess of 85% $\dot{V}O_{2\,max}$, the T_{re} is sensitive to changes in environmental conditions. During severe ex-

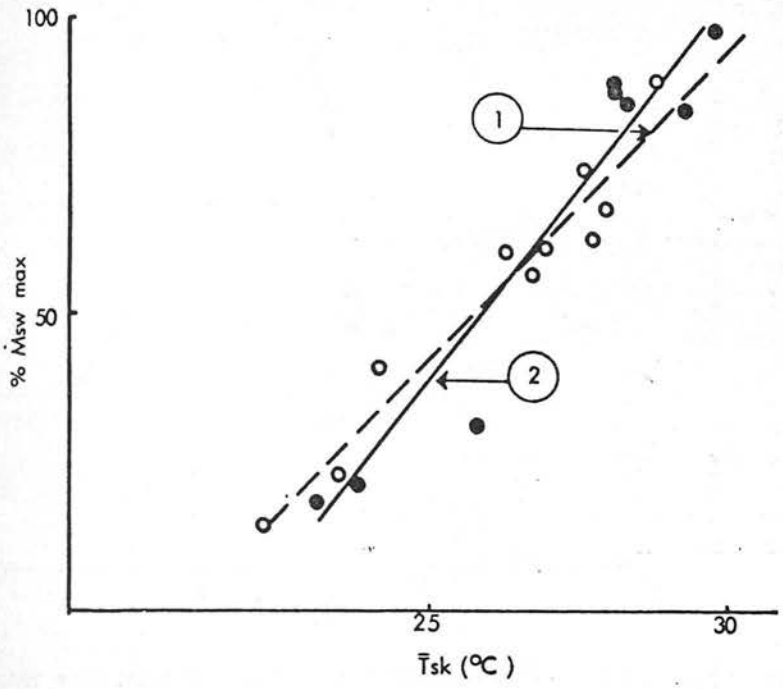

FIG. 6. Evaporative sweat loss expressed at a % of maximal evaporative sweat loss ($\%\dot{M}_{sw,max}$—see text) in relation to mean skin temperature ($\bar{T}_{sk}$). Linear regression lines shown are of the form $\%\dot{M}_{sw} = -213.9 + 10.27\ \bar{T}_{sk}$ (°C); $r = +0.97$ (*subj 1*); $\%\dot{M}_{sw} = -287.6 + 13.07\ \bar{T}_{sk}$ (°C); $r = +0.95$ (*subj 2*) exercising at 65% $\dot{V}O_{2\,max}$.

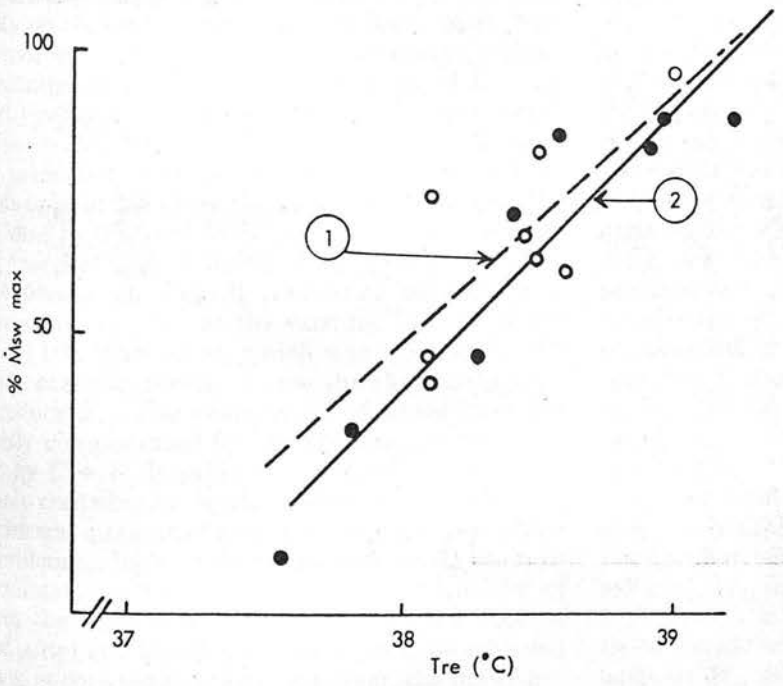

FIG. 7. Relative evaporative sweat loss ($\%\dot{M}_{sw,max}$) in relation to T_{re} at a given $\bar{T}_{sk}$ of approximately 28°C. T_{re} range 37 – 39.3°C see text. $\%\dot{M}_{sw} = -1583 + 42.96\ T_{re}$ (°C); $r = +0.73$ (*subj 1*) $\%\dot{M}_{sw} = -1875 + 50.39\ T_{re}$ (°C); $r = +0.93$ (*subj 2*).

ercise T_{re} rose as a linear function of time (Fig. 1) beyond the 20th min of work to reach values approaching and often exceeding 40°C even in moderate conditions (T_{db} = 21°C, rh <50%) with high convective airflow. Equally at T_{db} below 10°C T_{re} fell and values observed in the two subjects were similar to those recorded in moderate and warm environments at lower relative loads (Fig. 2). These findings are contrary to those of Nielsen (14) and several later authors (9, 13, 24), who have found the rise in T_{re} during exercise to be independent of ambient temperature from 5 to 30°C at lower relative work loads and less convective cooling.

In a previous paper (3), we have argued that at very high relative work loads that can be sustained for 1 h, the skin becomes partially vasoconstricted, resulting in a fall of $\bar{T}_{sk}$. This will inhibit sweating and though $\dot{M}_{sw}$ appears to reach a maximal value (Fig. 5) with apparent saturation of the sweat loss mechanism, we have argued that $\dot{M}_{sw}$ is not the limiting factor for thermal equilibrium under these conditions. The present observations serve to underline this view. The changes in $\bar{T}_{sk}$, which can be achieved in relation to the large increase in heat production, are small due to the high cooling capacity of the environment, thus one might expect (11, 13, 24), as has been found (Fig. 4), $\bar{T}_{sk}$ to be a linear function of T_{db}. However, it should be noted that the slope of the $\bar{T}_{sk}/T_{db}$ line is significantly steeper at 85% than 65% $\dot{V}O_{2\,max}$, which is indicative of a greater degree of vasoconstriction

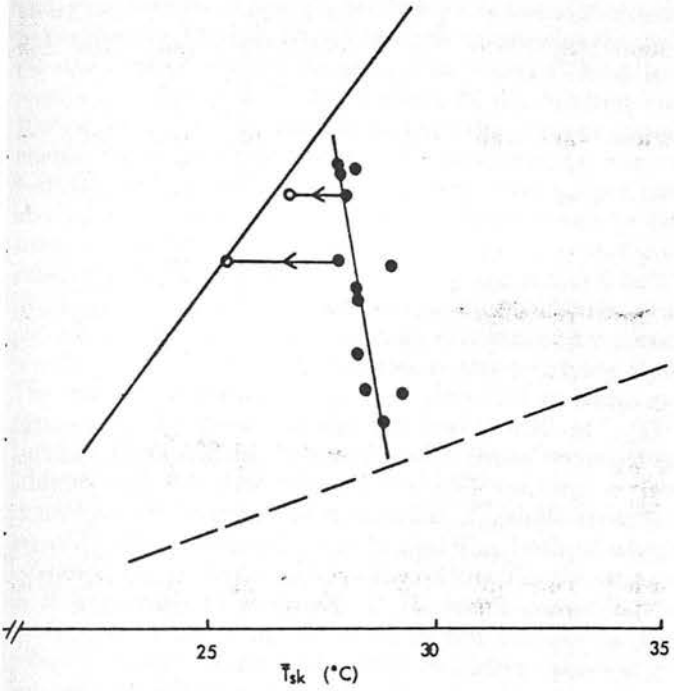

FIG. 8. Effect of skin wetting on $\bar{T}_{sk}$ in relation to heat dissipated from skin (H_{sk}) and total (——) cooling capacity of environment provided by evaporation and convection plus radiation ($C + R$ - - -). Values were calculated using heat exchange coefficients given by Kerslake (8), Appendix 4. (○) skin wetting, (●) normal data; *subj 2*.

igher work load. The degree of inhibition of $\dot{M}_{sw}$ work loads at a T_{db} of 21°C can be judged from ılts on *subject 1*, working at a lower work load environment sufficiently warm to ensure periph- odilatation, he achieved 30% increase in $\dot{M}_{sw}$ (1.5 l/h) compared with his severe work experiments derate environment. Thus the sweat mechanism s saturated in moderate environments at high ads only in the sense that a higher $\dot{M}_{sw}$ cannot be d due to the cool skin imposed by the require- f exercise. This is clearly illustrated by the skin- experiments (Fig. 8) performed on *subject 2*, sweating capacity at the existing $\bar{T}_{sk}$ was of the f 1.0 l/h. The water, which was sprayed on the ring exercise, served to cool the skin further but reduce T_{re}. The evaporation of water from the actly compensated for the reduction of $\dot{M}_{sw}$ and ss by $C + R$. It subjectively aided performance, only contribution to the achievement of thermal rium was qualitative rather than quantitative. The roblem at high levels of exercise would seem to maintenance of an adequate convective flow of om the core to the periphery. In the face of ed peripheral blood flow, this can only be achieved nges in core temperature. However, the disadvan- this mechanism as shown by Wyndham (25) is e T_{re} becomes very sensitive to small changes in ironment (Fig. 2). Due to the body's small thermal y, at high rates of heat production the changes in temperature are rapid. The subject becomes ef- ly poikilothermic.

ourse, the question may be raised as to why the ot allowed to rise during exercise when T_{re} reaches (Fig. 3 and 5). At high absolute levels of heat tion with a high T_{re} and low $\bar{T}_{sk}$, the rise in $\bar{T}_{sk}$ ed to effect a given heat transfer to the environ- is small. For instance, a rise in $\bar{T}_{sk}$ of only 1°C produce a substantial rise in $\dot{M}_{sw}$ (Fig. 6), and increased heat loss by $C + R$. The answer is probably as pointed out by Rowell et al. (21) that at high levels of work metabolic considerations are given priority over the body's requirements for heat dissipation. A rise in $\bar{T}_{sk}$ can only be achieved by an increase in skin blood flow at the expense of muscle blood flow. This would inevitably compromise the level of exercise being performed and ultimately result in a reduction in the speed of running. Certainly, it is our experience in the laboratory that at high work loads if one suddenly forces the skin to dilate using only small amounts of radiant heat, the subject's performance immediately suffers and he is unable to sustain the work. Similarly, outdoors it is a common observation of marathon runners (including *subject 1*) that they become extremely sensitive to small changes in environmental conditions during a race, particularly on sunny days when there is limited cloud cover and intermittent shade, which result in adjustments of pace.

At low environmental temperatures (T_{db} 5°C), one observes a similar, but essentially opposite, thermal behavior when severe work is performed. The $\bar{T}_{sk}$ is further reduced, $\dot{M}_{sw}$ inhibited, and T_{re} actually falls (Fig. 2). At 5°C the $\bar{T}_{sk}$ is of the order 21.5°C, though even under these conditions the large metabolic heat production sustains $\dot{M}_{sw}$ at approximately 40% of maximum (Fig. 6). The observation underlines the enormous potentiating effect of exercise on $\dot{M}_{sw}$ (19) and the inherent difficulty of suppressing sweat completely during work (24). From Fig. 6 it would seem that a $\bar{T}_{sk}$ of ~15.5°C would be required to totally inhibit sweating; this would prove unacceptable to most subjects and one doubts if one could achieve a physiological condition under which sweating ceased at very high levels of exercise. At the low T_{db}, K was higher at 5 than at 10°C, and though the skin was probably maximally vasoconstricted, there were greater regional variations in skin temperature than observed at higher T_{db}. Because one must assume that the requirements of the muscles for blood flow remain large

and constant during severe exercise, these observations would suggest that forced enlargement of the body's core under conditions of low T_{db} facilitate the transfer of heat by conduction directly from the working muscles through the overlying tissue to the skin. The "forced" heat loss results in a lowered T_{re} for a given M (cf. Nielsen and Davies, Ref. 15). The two subjects in the present experiments were extremely thin (the conventional sum of four-skinfold thicknesses was <25 mm), and indeed this may be one reason why they were able to complete the strenuous series of experiments. The high 'minimal' and relatively low 'maximal' K values recorded at 5 and 21°C, respectively, suggest that in both subjects a large proportion of the heat produced was eliminated by direct transfer from the working muscles to the overlying skin. The maximum mean values of K recorded in these experiments, 39.3 $W \cdot m^{-2}$ (*subject 2*) and 50.4 $W \cdot m^{-2} \cdot °C^{-1}$ (*subject 1*) should be compared with those recorded in athletes (16, 17) and nonathletes (26) working in hot chambers. Of course, the maximum K values recorded are critically associated with M and T_{db}, both of which influence $\bar{T}_{sk}$. At high work loads and low $\bar{T}_{sk}$, the change in K necessary to maintain T_{re} is small compared to conditions in the heat. Working in hot conditions, the present subjects could easily achieve higher values of K, but only at lower work loads. They could not achieve their highest work output for 1 h if $\bar{T}_{sk}$ exceeded ~28°C.

In the present experiments with high convective air flow, K was closely associated with $\dot{M}_{sw}$ at all levels of work. This suggests, if we accept the work of Nielsen (13), which shows K may be taken as an indication of peripheral blood flow, that the control of evaporative sweat loss and skin circulation are closely matched. Indeed, the common thermal input may be central core temperature (27). Clearly, if this is so, direct heat transfer not involving peripheral blood flow would be a distinct advantage for high performance. In our thin subjects, one would expect a higher $\dot{M}_{sw}$ for a given K than seen in normal subjects working in environments with lower airflows and there is some evidence of this (cf. *Eq. 2* with Kerslake (8), p. 169, and Fig. 7 of Ref. 22). The low K value and relatively high sweat rates at low $\bar{T}_{sk}$ (Fig. 6) during severe work suggest that sweating can be initiated and sustained at minimal peripheral blood flow levels. The magnitude and importance of direct heat transfer and the level of skin blood flow necessary to elicit a given sweat rate in subjects who vary in body size and composition would be an interesting field for further research.

The linear relationship of $\dot{M}_{sw}$ to K allows prediction of $\dot{M}_{sw}$ with a standard deviation of ± 63 $W \cdot m^{-2}$ in the two subjects. However, if we assume proportionality between $\dot{M}_{sw}$ and K, then for a given relative work load (and therefore T_{re}, Fig. 3) $\dot{M}_{sw}$ should be a curvilinear function of $\bar{T}_{sk}$ as Kerslake (8) has indicated. There is no evidence of this in Fig. 6; $\dot{M}_{sw}$ expressed as a $\%\dot{M}_{sw,\,max}$ is a linear function of T_{re} (Fig. 7). These observations find agreement with those (e.g., Ref. 11) who have stressed the importance of $\bar{T}_{sk}$ in sweat regulation and the extensive work of the Pierce Laboratory (11, 22) that has consistently advocated a model of sweating based on $\bar{T}_{sk}$ and T_{re} (or T_{es}). Their current model takes the form of a multiple regression equation with the addition of an exponential term to account for inhibition of sweating due to local skin cooling. The model has proved useful for predicting sweat rates in positive (12) and negative (10) work in subjects who have similar levels of $\dot{V}O_{2\,max}$ and states of acclimatization. The limitation of the equation without resort to complex and as yet unknown constants is in reconciling $\dot{M}_{sw}$ in subjects who differ in thermal status and fitness. The present analysis suggests that the natural state of acclimatization of subjects living and working in temperate climates may be a function of their exercise capacity (cf. Gisolfi and Robinson, Ref. 4). From the observed data, the following multiple regression equation can be derived

$$\%\dot{M}_{sw} = -1814 + 44.45\ T_{re}\ (°C) + 5.90\ (\bar{T}_{sk}\ (°C)); \qquad r = 0.82 \tag{2}$$

Using the equation, $\dot{M}_{sw}$ can be predicted with a standard deviation of ± 12%. If one inserts the set point constants of 36.9°C, i.e., allowing 0.2°C difference between T_{es} and T_{re} (Saltin and Hermansen (23)) for T_{re} and 34°C for $\bar{T}_{sk}$ used by Nadel et al. (11), the relationship of $\dot{M}_{sw}$ to $\bar{T}_{sk}$ and T_{re} is not materially changed. The constant terms are qualitatively of the same order of magnitude though the intercept becomes positive. As a T_{re} of 36.9°C is closely in agreement with our data (Fig. 7) for the set point of sweating with a $\bar{T}_{sk}$ of 28°C, the positive intercept may be in part due to the figure of 34°C given by Nadel et al. (11) for the skin with zero central drive. At rest there is some evidence to suggest that their figure is accurate (5), but during exercise, particularly with high convective air flow, the sensitivity of the skin thermoreceptors may change. Certainly, the present analysis would suggest that the figure is too high, but further detailed experiments would be required before definitive conclusions could be reached. The important factor that emerges from the analysis is that at a set $\bar{T}_{sk}$ a given increase in T_{re} will produce a set rise in relative sweat rate in both the nonathlete and the athlete. The aerobic power output and cooling capacity of the body are seemingly closely matched in both subjects and controlled in a similar way independently of the fitness of the individual. A rise in core temperature of 1°C in a moderate environment with airflow between 2.5–5 m/s will produce a 40% increase in sweat rate (Fig. 7). This effect will be modified by any change in $\bar{T}_{sk}$.

If these observations are taken together, then the rise in T_{re} is seen to meet the requirements of heat dissipation. Though the rise in T_{re} may be related to M, the rate of work and the relative work load, its primary functions are to create a thermal gradient from core to periphery and to elicit a given sweat rate response. Beyond $\dot{M}_{sw,\,max}$, T_{re} rises passively to maintain the convective transport of heat from the core to the skin in the face of an active increase in peripheral vasoconstrictor tone. Therefore, at all times during exercise T_{re} rises or falls to maintain a balance between heat production and heat loss. The environment exerts the greatest influence on $\bar{T}_{sk}$ and both the active and passive roles of $\bar{T}_{sk}$ on thermal regulation can be seen in Fig. 6 and 8. Changes in $\bar{T}_{sk}$ modify the central drive to $\dot{M}_{sw}$ (Fig. 6) and possibly peripheral blood flow (*Eq. 2*). Certainly local temperature

most important factor in the control of blood flow in superficial vessels of the skin (1, 7). The passive role ... is exemplified by the skin-wetting experiments ... the subject responds in a manner similar to a ...-bulb thermometer (Fig. 8). However, it is clear that ...gh metabolic rates the intense sympathetic outflow ...erride thermal stimuli (2, 20) and produce a fall in ... Fig. 4). Performance at these levels of exercise will ...ed on $\dot{V}O_{2\,max}$, the maintenance of a low $\bar{T}_{sk}$ (at or ... 28°C), and the cooling capacity of the environ-... On this basis, given that $\dot{V}O_2$ is a linear function of ...ed (18) and knowing the $\dot{V}O_{2\,max}$ of an endurance athlete and the environmental heat-transfer coefficients, it should be possible with the aid of plots similar to Fig. 8 to predict optimal performance for different climatic conditions.

I thank Miss Jane Allen for her technical assistance and Martin Thompson for his willing cooperation as a subject for these experiments. I am deeply indebted to Dr. Griffith Pugh for many hours of discussion regarding some of the ideas contained in this manuscript. It was he who first suggested the approach shown in Fig. 8 for predicting optimal performance of running under different environmental conditions.

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The effects of atropine and practolol on the perception of exertion during treadmill exercise

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The role of heart rate (f_H) in the perception of exertion (RPE) during short term progressive and prolonged (1 h) exercise has been studied in healthy male subjects by observing the effects of experimentally changing f_H, using intravenous injections of either atropine or practolol to modify the influence of the autonomic system.

In short term exercise the results showed that independently of the large changes produced in f_H for a given oxygen intake, the subjects rated their exertion in accordance with the relative work performed (*i.e.* $\%\ \dot{V}_{O_2max}$) in all experiments with and without drug administration. In prolonged exercise the association of RPE and $\%\ \dot{V}_{O_2max}$ was less clear. After 10 min of prolonged exercise at the same $\dot{V}_{O_2}$ the RPE scores were lower than those observed in the progressive work tests and with atropinisation and β-blockade RPE tended to rise during the exercise to reach a maximum value at the 60th min though $\dot{V}_{O_2}$ remained unchanged.

It was concluded that heart rate *per se* has little influence on RPE and is not an important factor underlying the perception of effort. Nor can it be used to explain the close association between RPE and relative work load found in short term exercise. During prolonged exercise more research is required before any definitive conclusion can be reached particularly bearing in mind the importance of RPE scales to the study of industrial and occupational stress.

1. Introduction

The scale suggested by Borg (1962, 1970) for the rating of perceived exertion (RPE) is constructed to give a linear increase with heart rate when normal cycling is performed on a bicycle ergometer. From previous work in our laboratory (Sargeant and Davies 1973, 1976) and others (Edwards *et al.* 1972, Henriksson *et al.* 1972) it is clear that the relationship between heart rate and RPE is indirect and must represent the integration of a number of physiological factors which enable the level of exertion to be accurately perceived in relative terms so that regardless of the type of exercise and the $\dot{V}_{O_2max}$ attainable there remains a strong association between relative work load (expressed as $\%\ \dot{V}_{O_2max}$) and RPE.

It is not, however, clear which factors are implicated in this integration and in order to examine more specifically the role of heart rate in the perception of exertion we have studied healthy male subjects performing progressive short term exercise and prolonged exercise on a treadmill under control conditions and following injection of either atropine or practolol. The primary effect of these drugs is to modify heart rate response for a given oxygen intake (Ekblom and Goldberg 1971) or relative work load by reducing vagal and sympathetic tone respectively.

2. Methods

Four healthy male subjects performed progressive short term exercise walking at constant speed on a motor driven treadmill under control conditions and after intravenous injection of atropine (1·8 mg) and practolol (15 mg). Starting at zero the treadmill gradient was increased by 3% every 8 min until subjects reached exhaustion. During the last two minutes at each gradient, heart rate (f_H) was recorded and oxygen uptake ($\dot{V}_{O_2}$) determined by the open circuit (Douglas bag) technique. The CO_2 and O_2

concentrations were measured using physical gas analysers which were calibrated at frequent intervals with gas of known concentration. Rated perceived exertion (RPE) was assessed using the revised scale proposed by Borg (1970) giving scale values 6–20, each odd number of the scale being accompanied by a brief verbal description as shown below. Subjects were shown the chart at the end of each work load and asked to indicate a number on the scale corresponding to the degree of exertion perceived.

6
7 very, very light
8
9 very light
10
11 fairly light
12
13 somewhat hard
14
15 hard
16
17 very hard
18
19 very, very hard
20

In addition, data were collected during prolonged exercise under control conditions and after intravenous injection of either atropine or practolol from six subjects (two of whom performed the progressive exercise). This consisted of walking on the treadmill for 1 h at constant gradient and speed chosen to correspond to approximately 60% of the subject's maximum oxygen uptake.

At approximately 10, 25, 45 and 60 min of exercise, f_H, $\dot{V}_{O_2}$ and RPE were determined as previously described. In addition, blood samples were taken for the estimation of lactate concentration (Gercken 1960), rectal temperature was monitored from a thermistor probe, and average skin temperature assessed from the weighted temperatures of 13 sites on the body after the method of Hardy and Dubois (1938).

Since Ekblom *et al.* (1972) have shown that atropine and β-blockade have no effect on maximal aerobic power output ($\dot{V}_{O_2max}$), the $\dot{V}_{O_2max}$ was measured in separate control experiments on the treadmill using the Douglas bag technique. The criteria for maximal performance were that $\dot{V}_{O_2}$ showed no further increase with increasing work load and plateau values of $\dot{V}_{O_2}$ agreed within $\pm 5\%$ (Davies 1968). None of the subjects had previously taken part in experiments using atropine or practolol, and they were given no information as to the drug to be administered before the start of exercise.

3. Results

3.1. *Short term progressive exercise*

At a $\dot{V}_{O_2}$ of $2\,l\,min^{-1}$, practolol caused a significant (30 beats min^{-1} depression of heart rate whilst atropine caused an elevation (~ 10 beats min^{-1}) when compared with exercise performed under control conditions. $\dot{V}_{O_2}$ for a given work load was unaffected by the administration of the drugs.

During progressive short term exercise the relationship of perceived exertion (RPE) to heart rate f_H) was displaced to the right by atropine and to the left by practolol (figure 1).

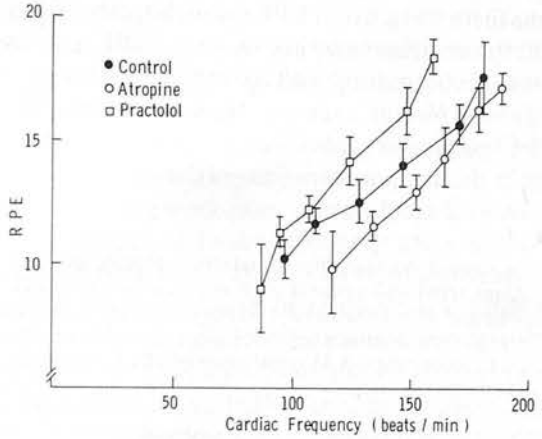

Figure 1. RPE in progressive exercise, plotted against cardiac frequency. Mean (±SD) data for each treadmill gradient are shown for control conditions (●): and after intravenous injections of atropine (○), and practolol (□).

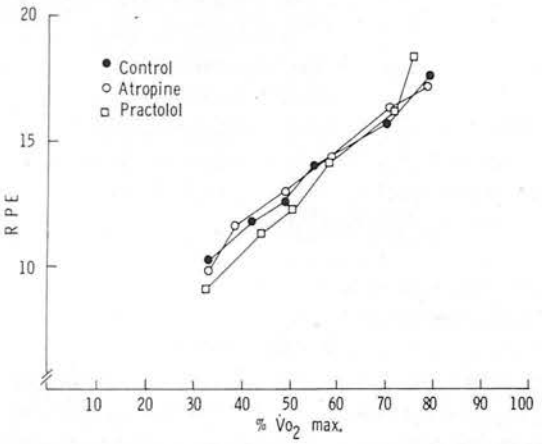

Figure 2. RPE in progressive exercise, plotted against $\%\dot{V}_{O_2max}$.

However, when perceived exertion was related to the relative work load for each subject ($\%\ \dot{V}_{O_2max}$), the differences between the control and drug conditions disappeared. The relationship is given by:

$$\text{RPE} = 5{\cdot}59 + 0{\cdot}15\ (\%\ \dot{V}_{O_2max}),\ r = 0{\cdot}94,\ \text{SD} = 1\ \text{RPE (Figure 2)}.$$

3.2. *Prolonged exercise*

After 10 min prolonged exercise at 60% $\dot{V}_{O_2max}$ mean RPE scores were 12·3, 12·2, and 12·7 respectively for control, atropine and practolol experiments (table 1). This is in contrast to, and substantially lower than the RPE score of 14·6 at 60% $\dot{V}_{O_2max}$ derived from the observed relationship in short term progressive exercise.

Under control conditions, RPE rose only slightly (mean 12·3 to 12·9 RPE) during the 60 min of exercise, although both f_H and rectal temperature increased significantly ($+14$ beats min^{-1} and $+1{\cdot}1°C$ respectively). After intravenous injection of practolol, RPE scores were similar to the control values at 10 min despite a lower f_H (-16 beats min^{-1}). RPE scores were also similar after 10 min exercise in the atropine experiments where f_H and mean skin temperature was elevated ($+18$ beats min^{-1} and $+1{\cdot}6°C$). In

both drug conditions there was a rise in RPE throughout the 60 min of exercise, but this was most marked in the atropine experiments where RPE rose from 12·2 at 10 min to 15·2 RPE after 60 min. In comparison with the control conditions where there was little change in RPE during 60 min exercise, there were greater increases in rectal temperature (+1·5°C compared with a rise of 1·1°C) and more notably mean skin temperature so that in the atropine experiments the final value was 35·0°C (compared with 31·1°C under control conditions).

Table 1. Physiological responses after 10 and 60 min of prolonged exercise performed at constant speed and gradient on a motor driven treadmill under control conditions and after intravenous injections of atropine and practolol. Mean (±SD) are given for relative work load (% $\dot{V}_{O_2\,max}$), RPE, heart rate (f_H), plasma lactic acid concentration (LA), rectal temperature (T_{re}) and mean skin temperature ($\bar{T}_{sk}$).

	Control		Atropine		Practolol	
	10 min	60 min	10 min	60 min	10 min	60 min
% $\dot{V}_{O_2\,max}$	60	64	60	63	62	62
	±7	±8	±8	±6	±9	±7
RPE	12·3	12·9	12·2	15·2	12·7	14·3
	±1·3	±1·3	±1·3	±2·2	±1·2	±1·8
f_H(beats min^{-1})	150	164	168	185	134	147
	±17	±13	±12	±13	±18	±14
LA(mM 100 ml^{-1})	1·7	1·6	1·7	2·9	2·1*	1·9*
	±0·9	±1·0	±0·8	±1·9	—	—
T_{re} (°C)	37·4	38·5	37·4	38·9	37·3	38·8
	±0·4	±0·4	±0·4	±0·5	±0·6	±0·4
$\bar{T}_s^k$ (°C)	30·7	31·1	32·3	35·0	29·7	29·9
	±0·7	±0·8	±1·4	±0·7	±0·7	±0·8

*$n=3$

4. Discussion

In a previous paper (Sargeant and Davies 1973) we demonstrated how the relationship of RPE with heart rate could be modified in the same group of subjects by having them perform different types of ergometer exercise involving cranking with one or two arms and pedalling with one or two legs. Although this clearly indicated that heart rate cannot be the sole factor responsible for the perception of exertion it could still be argued that it does play a differential role in combination with other changes occurring in response to exercise.

The present paper sought to examine this possibility by experimentally altering the heart rate response for a given work load during treadmill walking. This was achieved by modifying the influence of the autonomic nervous system with either atropine or practolol, neither of which in the dosage used altered the $\dot{V}_{O_2max}$ or $^{\infty}{}_{O_2}$ for a given work load (Ekblom *et al.* 1972, and Davies *et al.* 1978). In our experiments each subject performed an identical progressive exercise test under the three experimental conditions so that the movement sensations arising from joint and muscle receptors were the same; but as can be seen (figure 1) the heart rate/perceived exertion relationships were quite different in the three experimental conditions. These data are consistent with the findings of Ekblom and Golbarg (1971). When the RPE was related to relative work load (% $\dot{V}_{O_2max}$) these differences disappeared. Thus during progressive short term work the subjects were able to identify correctly the relative intensity of a work load during exercise on a motor driven treadmill and were uninfluenced by the large variations in f_H resulting from the administration of atropine and practolol.

4.1. *Prolonged exercise*

The relationship of RPE to $\% \dot{V}_{O_2max}$ during prolonged steady state exercise is less clear. At a given $\% \dot{V}_{O_2max}$ RPE scores after 10 min prolonged exercise were lower than those scored during progressive exercise.

The difference may arise from the experimental design used for progressive and prolonged exercise. RPE scores were taken in prolonged exercise at a relative work rate of $\sim 60\% \dot{V}_{O_2max}$. At corresponding work rates in progressive exercise, RPE scores were taken after the subjects had performed a series (three or four) of increasing work loads whose cumulative effect may have been to raise RPE. Unfortunately we were unable to examine this effect due to the small range of relative work loads performed in the prolonged exercise tests. It should, however, be pointed out that other investigators (*e.g.* Stanford 1975 have looked at this problem and have failed to identify any such cumulative effect in progressive exercise. In the two subjects who performed both forms of exercise, differences in RPE at a given $\% \dot{V}_{O_2max}$ could not be related to differences in ventilation, cardiac frequency or lactate between the two forms of work. However, it is possible that the knowledge of the type of exercise to be performed by each subject could have altered the subjective readiness and perception of the exercise, as some reports have indicated that RPE can be altered by the character traits of the subject (Morgan 1973) and by psychological suggestion (Morgan *et al.* 1973).

Over the 60 min period of prolonged exercise there were no clear relationships between several physiological measurements and RPE (table 1). The somewhat higher RPE scores for drug experiments at 60 min may reflect impairment of cardiovascular and thermoregulatory function. However, if so, such impairment was not definitively reflected by any single measurement of physiological change, and neither did multiple regression analysis indicate any significant association.

The increase under all conditions in the perceived level of exertion over 60 min may reflect a reduction in the maximal working capacity in subjects performing prolonged work: Such reduction may be a result of the increasing demand placed on the circulation by thermoregulatory needs in competition with metabolic needs. However, it is worth noting that in some atropine experiments where subjects were unable to continue exercise, apparently due to an inability to thermoregulate as indicated by high skin temperatures, the final RPE scores were still relatively low suggesting that some direct work related factor was dominating the perceptual response.

Thus, our data indicates that heart rate *per se* is not an important cue for the perception of exertion and does not appear to make a significant contribution to the close association between RPE and the relative work load performed by the individual in short term exercise. In prolonged exercise the position is less certain. One important problem which needs to be resolved is the apparent anomaly between the scoring of perceived exertion during progressive compared with prolonged exercise at the same relative work load. Clearly further data is needed especially in view of the potentially important practical application of RPE scales to the study of occupational stress.

Le rôle de la fréquence cardiaque (F_c) dans la perception de l'effort (R.P.E.) au cours d'un exercice progressif court ou prolongé (1 heure) a été étudié chez des sujets masculins en bonne santé, en observant les effets d'une variation provoquée expérimentalement dans la F_c au moyen d' injections d'atropine ou de practolol, agissant sur le système autonome. Au cours de l'exercice à court-terme, les résultats montrent, qu'indépendemment des fortes variations dans la F_c pour une consommation d'oxygène donnée, les sujets jugent leur effort en fonction du travail relatif effectué (c.à.d. le $\% \dot{V}_{O_2max}$) dans toutes les expériences avec et sans administration des drogues. Au cours de l'exercice prolongé, la relation entre la R.P.E. et $\% \dot{V}_{O_2max}$ était moins claire, Après 10 mn d'exercice prolongé, pour la même $\dot{V}_{O_2}$, les scores R.P.E. étaient plus bas que ceux observés au cours des tests de travail progressif. Avec l'atropinisation et le blockage-β, la R.P.E. tendait à

s'élever pendant l'exercice pour atteindre une valeur maximum à la 60e minute, bien que la $\dot{V}_{O_2}$ restât inchangée. On peut en conclure que la fréquence cardiaque, en soi, a peu d'influence sur la R.P.E. et ne constitute pas un facteur important pour la perception de l'effort. Elle ne peut guère servir à expliquer la relation importante entre la R.P.E. et la charge de travail relative qui est apparue au cours de l'exercise à court-terme. En ce qui concerne le travail prolongé, il semble que d'avantage de recherches soient nécessaires avant de parvenir à une conclusion définitive tout en tenant compte de l'importance des échelles de R.P.E. pour l'évaluation des contraintes industrielles et professionnelles.

Während kurzdauernder progressiver und langdauernder (1 h) körperlicher Belastung wurde die Bedeutung der Herzschlagfrequenz (f_H) für die Anstrengungsempfindung (AE) bei gesunden männlichen Probanden untersucht. Dabei wurde der Einfluß des autonomen Nervensystems auf f_H durch intravenöse Injektionen von Atropin und Practolol systematisch variiert.

In den Kurzzeitversuchen stuften die Probanden ihre Anstrengung unabhängig von der medikamenten-induzierten Änderung von f_H entsprechend ihrer relativen Belastung (d.h. % der Leistung bei $\dot{V}_{O_2max}$) ein. In den Langzeitversuchen war der Zusammenhang zwischen der AE und der relativen Leistung weniger deutlich. Nach 10 Minuten der Langzeitbelastung bei einer konstanten Sauerstoffaufnahme lag die AE-Einstufung niedriger als während der progressiven Kurzzeitbelastung. Unter Medikamentengabe zeigte die AE-Einstufung während der Belastung einen ansteigenden Trend mit einem Höchstwert in der 60. Minute trotz konstanter Sauerstoffaufnahme.

Es wird geschlossen, daß die Herzschlagfrequenz als solche keine wesentliche Rolle bei der Anstrengungsempfindung spielt. Sie erklärt auch nicht die enge Beziehung zwischen AE-Einstufung und relativer Belastung in den Kurzzeitversuchen. Im Hinblick auf die Wichtigkeit der AE-Skalierung bei Arbeitsplatzuntersuchungen müssen weitere Langzeitversuche durchgeführt werden, bevor endgültige Aussagen gemacht werden können.

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Temperature Regulation in Anorexia Nervosa Patients during Prolonged Exercise

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ABSTRACT. The thermal responses to prolonged exercise on a stationary bicycle ergometer have been studied in 10 anorexia nervosa (AN) patients and 5 normal subjects. The patients were young (12–18 years) females (except for 1 case) and had less than 10% of their body weight as fat. The basic experiments were conducted in a moderate environment (24°C) at approximately 65% maximal aerobic power output on all patients and controls. In addition, 2 patients were studied in warm (32°C) conditions, 1 patient in cool (12°C) conditions and 2 patients after being heated for 30 min in a sauna at a temperature of approximately 50°C. Measurements included oxygen uptake, metabolic (M) and total heat production, evaporative sweat loss (E), rectal (T_{re}) and mean skin temperatures. The results showed that T_{re} (for a given M) was higher and E lower in the patients than the controls. Passively heating the anorexia patients before exercise increased resting T_{re} and E but did not affect the "plateau" value of T_{re} obtained at the end of work. In the warm environment, T_{re} rose to the same level as observed at 24°C but did not rise above the resting value in cool conditions. Thus it would appear that although patients with AN can regulate their body temperature adequately in a moderate environment, loss of body fat which reduces thermal insulation may decrease the range of ambient temperatures over which T_{re} can be maintained during exercise. Our data do not support the theory of a loss of central (hypothalamic) thermoregulatory control in anorexia.

Key words: anorexia nervosa, temperature regulation, prolonged exercise, rectal temperature, skin temperature, sweat loss.

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Primary anorexia nervosa (AN) is universally characterized by weight loss due to a failure to eat (3, 5). The physical features of AN are those of chronic inanition; the patients are often extremely emaciated with pallid and sallow skin but rarely show signs of vitamin, iron or protein deficiency diseases. One of the major physical concerns of the patients is a feeling of cold, particularly in the extremities. Their hands and feet even in a thermally neutral environment are vasoconstricted, acrocyanosed and give rise to a feeling of numbness and sometimes pain. It is surprising therefore that although low body temperatures and hypothermia (22) have often been reported in AN, few attempts have been made to study thermoregulation in this disease. Wakeling and Russel (21) have reported the effects of a localized peripheral heat stress, induced by placing the arm up to the elbow in water at 45°C, on oral and finger temperatures in 11 anorexic girls. Gleeson and Moore (10) have studied the effects of mild heat and Mecklenburg et al. (14) of cold stress in a climatic chamber on 5 anorexia nervosa patients.

To our knowledge, no attempt has been made to study the thermoregulatory responses to a given metabolic heat load resulting from exercise in AN. The present investigation examines metabolic and thermal responses to prolonged work in 10 patients with AN and 5 healthy normal subjects.

SUBJECTS AND METHODS

The patients formed part of a larger study (8, 9). They conformed to the following criteria for AN, chiefly accord-

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Abbreviations: AN=anorexia nervosa, $\dot{V}_{O_2}$=oxygen uptake, $\dot{V}_{O_2}$ max = maximal aerobic power, T_{re} = rectal temperature, T_{sk} = skin temperature, $\bar{T}$ = mean temperature, M = metabolic heat production, H = total heat production, E = evaporative sweat loss, S = heat storage, T_{db} = dry temperature, T_{wb} = wet temperature, C = convection, R = radiation.

Table I. *Physical characteristics of the patients and controls (mean ± 1 S.D.)*

Ht=height, Wt=weight, LBM=lean body mass

	Age (y.)	Ht (cm)	Wt (kg)	LBM (kg)	$\dot{V}_{O_2}$ max (l/min)
Patients (n=10)	15.0 ±2.0	164 ±15	37.9 ±8.1	34.1 ±7.2	1.18 ±0.30
Controls (n=5)	14.9 ±3.1	166 ±14	48.9 ±12.9		2.30 ±0.47

ing to Dally (5): 1) Age at onset less than 25 years. 2) Active refusal to eat with accompanying pronounced weight loss. 3) No evidence of schizophrenia, severe depression or organic diseases.

The physical characteristics of the patients with AN and the healthy controls are given in Table I. The anorexic state of the patients had lasted on average for 1.0 year (range 0.5–3) prior to the investigation. The normal menstruation and ovulatory cycles had ceased in the female patients at the time of measurement. They were studied at rest and during work on an electrically braked bicycle ergometer at approximately 65% of their maximal aerobic power ($\dot{V}_{O_2}$ max) for 1 hour. The subjects were weighed nude before and immediately after exercise, on a balance accurate to ±10 g. Rectal temperature (T_{re}) was measured during exercise from a thermocouple inserted into the rectum 8 cm above the internal sphincter. Skin temperature (T_{sk}) was measured at 14 sites, finger and thumb, hand, upper and lower arm, forehead, pectoral, sternum, abdomen, scapular, lumbar, anterior and posterior thigh and anterior and posterior calf, at rest and during the 2nd, 8th, 20th, 30th, 40th, 50th and 60th min of exercise using a thermistor probe mounted on an applicator. The recordings (except finger and thumb measurement) were weighted after the method of Hardy and Dubois (11), and the average was taken as the mean body skin temperature ($\bar{T}_{sk}$). Oxygen uptake ($\dot{V}_{O_2}$) was measured at rest and during the 28–30th and 58–60th min of exercise by the open circuit (Douglas bag) technique, O_2 and CO_2 content of expired air being determined by the micro-Scholander technique. Heart rate was calculated from standard ECG recordings at rest and every 2 min during exercise. From the raw data, calculations of metabolic (M) and total heat (H) production, evaporative sweat loss (E) and heat storage (S) were made using standard equations (15). Dry (T_{db}) and wet (T_{wb}) temperatures in the laboratory were 23.6±0.84 and 14.1±2.2°C, respectively. In two patients the experiments were repeated with and without preheating. For this purpose the patients sat in a sauna for 30 min at about 50°C T_{db} and 26°C T_{wb} prior to exercise. In addition, two patients performed exercise at an elevated environmental temperature (T_{db} 32.2±0.4, T_{wb}=17.6±1.40°C) and one patient worked in a cool environment (T_{db} 11.7±0.4, T_{wb} 6.4±0.5°C). Control experiments were conducted on 4 healthy boys and one healthy girl (Table I) in the laboratory and 2 healthy boys in the cool and warm environments.

$\dot{V}_{O_2}$ max was measured in the patients and control subjects on separate occasions. For reasons previously given and discussed (8) it was impossible to establish with abso-

Table II. *Thermoregulatory responses to exercise of AN patients and controls (mean ± S.D.)*

	$\dot{V}_{O_2}$ (l/min)	$\dot{V}_{O_2}$ max (%)	Load (W)	M (W)	H (W)	T_{re}[a] (°C)	$\bar{T}_{sk}$[a] (°C)	T_{re}[b] (°C)	$\bar{T}_{sk}$[b] (°C)	E (W)
12°C										
Patients (n=1)	1.31	73	90	447	357	36.58	30.09	36.80	29.10	3
Controls (n=2)	1.88	58	140	641	508	37.10	30.98	38.40	28.94	153
24°C										
Patients (n=10)	0.78 ±0.20	66 ±10	54 ±17	270 ±66	215 ±50	36.60 ±0.56	31.68 ±0.73	38.07 ±0.51	32.18 ±1.15	54 ±35
Controls (n=5)	1.61*** ±0.39	64 ±8	116*** ±32	548*** ±132	432 ±100	37.22** ±0.19	32.11 ±0.64	38.36 ±0.36	31.30 ±0.61	267*** ±69
24°C with preheating										
Patients (n=2)	1.07	68	80	363	298	36.75	33.43	38.40	32.60	112
32°C										
Patients (n=2)	0.91	56	75	311	236	36.55	33.19	38.25	35.84	136
Controls (n=2)	1.78	62	123	607	484	37.70	33.75	38.40	33.46	415

***p<0.001, **p<0.01.

[a] At rest. [b] At 60th min of exercise.

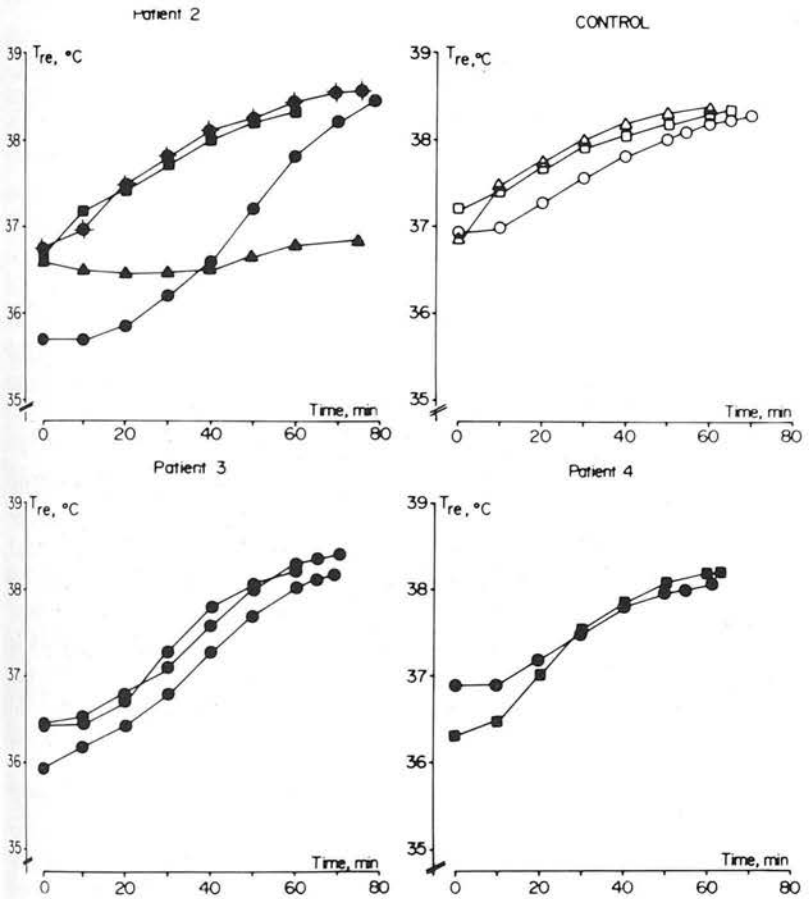

Fig. 1. Time course of T_{re} changes during exercise in 3 AN patients and 1 control subject at 12°C (▲, △), 24°C with (●) and without (●, ○) preheating; and at 32°C (■, □). Patient 2 developed signs of heat pyrexia at T_{re} 32°C and the exercise was terminated. It should be noted that the absolute level of T_{re} reached at the end of exercise in the patients was unrelated to the initial value and the change in body heat stores (cf. Bittel & Henane (4)) produced by preheating.

lute certainty the accepted criterion of $\dot{V}_{O_2}$ max, namely a plateau of $\dot{V}_{O_2}$ with increasing work load (6). Secondary criteria based on the work of Åstrand (1) were therefore used in this study. Evidence of maximal effort was accepted only if blood lactate exceeded 9 mmol/l and the respiratory exchange ratio was higher than unity. This was achieved in all patients and controls. Lean body mass was calculated from the sum of ten skinfold thicknesses after the method of Pařízková (17).

RESULTS

The metabolic and thermal responses to exercise of the anorexic patients and control subjects are given in Table II. Fig. 1 shows the typical T_{re} changes with exercise in AN in cool, moderate (with and without preheating) and warm environments. In moderate conditions without preheating, the T_{re} of the patient increases slowly, to reach a delayed "plateau" at 50th–80th min of exercise. Preheating raises the resting T_{re}, but has little effect on the rate of change and the final "plateau" level of T_{re} during exercise. Raising the ambient temperature to 32°C during the whole work period has the same effect; the onset of the T_{re} rise was more rapid but the rate of change and final value of T_{re} during exercise are similar to those found in the moderate environ-

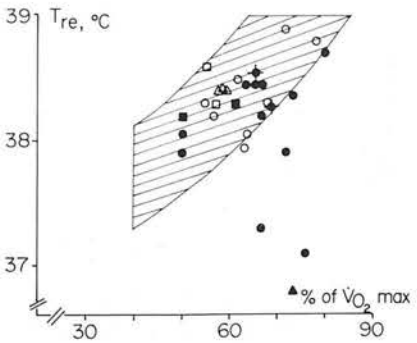

Fig. 2. Relationship of T_{re} to % $\dot{V}_{O_2}$ max. The shaded area shows the limits previously found for sedentary healthy subjects (7). Symbols as in Fig. 1.

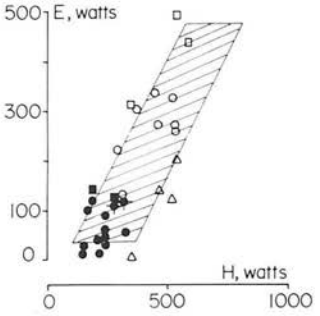

Fig. 3. E corrected for metabolic and respiratory water losses in relation to H during exercise. Symbols and shaded area as in Figs. 1 and 2.

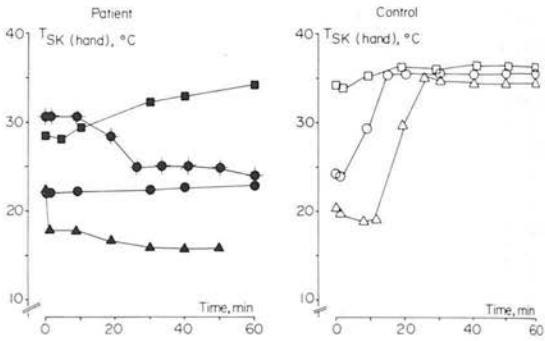

Fig. 4. Hand temperatures of an AN patient and a control subject exercising at 3 different environmental temperatures. Symbols as in Fig. 1.

ment. When the ambient temperature was decreased to 12°C, however, T_{re} did not rise above resting condition and remained at an almost constant level through exercise.

In the control experiments the rate of change and final plateau level of T_{re} were unaffected by ambient temperature over the range studied (Fig. 1 and Table II). At 24 and 32°C, T_{re} of patients and controls was related to M but more closely associated with the relative metabolic heat stress resulting from exercise (Fig. 2). The relationship of T_{re} to % $\dot{V}_{O_2}$max in 7 patients was similar to that found previously in healthy normal adults (7), but the points in 3 patients were below expected limits for the two variables (Fig. 2).

Absolute E was lower in the patients than in controls (Table II), but for a given H, E lay within the expected levels for normal subjects (Fig. 3). At 24°C the patients stored 10% of their H and dissipated 25% by evaporative and 65% by non-evaporative channels. The corresponding figures for controls were 7, 62 and 21%, respectively. Heat loss from the lungs corresponded to 9% of H in both groups. Preheating increased E by approximately 11% and decreased S to approximately 2% in two patients.

At the end of exercise at 24°C, $\bar{T}_{sk}$ was 0.88°C higher in the anorexic patients than in controls; preheating in the two patients had little effect on their final $\bar{T}_{sk}$ values. Increasing environmental temperature to 32°C raised E and $\bar{T}_{sk}$ and reduced the amount of heat dissipated by convection (C) and radiation (R) in both groups (Table II). However, although T_{sk} was higher in the anorexic patients at both environmental temperatures, there were distinct differences in the pattern of T_{sk} response to exercise in different parts of the body, particularly the extremities. For example (Fig. 4) the hands of the anorexic patients tended to remain cold and vasoconstricted even following preheating in moderate environment despite the fact that their $\bar{T}_{sk}$ remained higher than the controls'. Only when the patients exercised under warm conditions did the blood vessels of the extremities become dilated and T_{sk} of the hand rose to control values (Fig. 4).

In the anorexic patients who exercised at 12°C, E was suppressed completely, $\bar{T}_{sk}$ was similar to the controls' value, but again the temperature of the hand decreased to approximately 16°C.

DISCUSSION

In a moderate environment, AN patients appear to regulate their body (core) temperature in a way similar to that found in extremely sedentary and unacclimatized subjects (23). At rest the anorexia patients have low T_{re} (Table II) and body heat content, this gives rise to a greater capacity for S and delayed increase of T_{re} observed at the onset of work. During exercise at a dry bulb temperature of 24°C they have a higher T_{re} for a given M than control subjects but E for a given H was within the expected range previously found for normal subjects (Table II and Fig. 3). Passively heating the patients before exercise raised resting T_{re} and increased E, but it did not effect the rate of change in T_{re} during exercise nor the final plateau value of T_{re} at the end of work. In relation to relative work load, the T_{re} at the end of exercise lie within the lower

range of values previously found for normal subjects (7, 18).

A slightly reduced T_{re} for a given % $\dot{V}_{O_2}$ max might suggest that the loss of thermal insulation in the form of body fat may have a small effect on thermoregulation in AN. However, some caution is necessary in interpreting the results. We have alluded to the difficulties of measuring $\dot{V}_{O_2}$ max directly in AN patients (8). In normal subjects a slight bias in $\dot{V}_{O_2}$ max may be less critical, but in patients who have an extremely low $\dot{V}_{O_2}$, as in AN, it has a decisive effect. For example, an underestimation of (say) 200 ml/min^{-1} in a patient with $\dot{V}_{O_2}$ max of 1.5 l/min^{-1}, will result in an error of at least 10% in the calculation of relative work load. This factor could easily account for the small differences in, and the larger intersubject variability of, T_{re} for a given $\dot{V}_{O_2}$ max in 7 of the 10 patients studied compared with normal subjects (Fig. 2), but it could not explain the data on the remaining three patients exercising at 24°C, nor the results on the patient at 12°C. Undoubtedly a severe loss of body fat in AN will facilitate the convective transfer of heat from the core to the skin surface and it was observed that a greater proportion of H of AN patients, even in a moderate environment, is dissipated by convection and radiation ($C+R$). However, without knowledge of minimal conductance values in air for these anorexic patients, it is impossible to decide whether part of this heat loss by $C+R$ is obligatory due to their reduced thermal insulation or a result of active physiological regulation. A reduction in the amount of heat to be actively regulated would be expected to result in a lower T_{re} for a given M expressed as % $\dot{V}_{O_2}$ max, and ultimately to have a decisive effect on thermoregulatoin (16).

In cool conditions (12°C) the patient, despite the increase in M during exercise by shivering (Table II), was barely able to maintain his body temperature above resting conditions. The response by shivering in cold environment is contrary to reports by others (20). Sweating was suppressed and the heat loss from the body was solely due to respiration and $C+R$. In the control subjects T_{re} rose to the same plateau value as observed at 24°C, though E was reduced and $C+R$ increased. This suggests that the loss of body fat in anorexia may effect a reduction in the "prescriptive" (12, 13) range of ambient temperatures through which body temperature can be maintained and physiologically regulated. This is clearly an area for further research.

In warm conditions the thermoregulatory problems for the anorexic patient are the reverse of those encountered in a cool environment, for though the loss of fat facilitates the convective transfer of heat from core to skin, the raised ambient temperature reduces $C+R$ losses to the environment. In order to maintain thermal equilibrium the anorexic patient must sweat. They are clearly capable of doing this (Table II and Fig. 3); at 32°C, E increased by 64% during exercise in 2 patients over the value observed at 24°C. However, the maximal sweating capacity of the anorexic patient seems to be low. For example, during exercise at 32°C, one of the two patients studied showed signs of heat exhaustion and developed a pyrexia rash and the experiment had to be terminated before the 60th min at a T_{re} of 38.2°C (pat. 2, Fig. 1). None of the control subjects showed such signs when exercised under similar conditions.

Thus, anorexic patients appear to differ from normal subjects in their low sweating capacity and in the proportions of heat lost from the skin by evaporative and non-evaporative channels. In a moderate environment they behave, from the thermoregulatory point of view, in a way qualitatively similar to normal subjects exercising in a cool environment. This they appear to achieve to some degree by selective control of vasoconstrictor tone, particularly in the extremities, for although the anorexic patient maintains a slightly higher $\bar{T}_{sk}$ than controls (Table II), the T_{sk} of their limbs is extremely low (Fig. 4). Presumably in an attempt to conserve and control the amount of heat lost from the body surface, by $C+R$, the patient limits the size of his "core" by maintaining active vasoconstrictor tone in his extremities (2, 19). Under these conditions it has been shown (2, 19) that heat can be directly exchanged by a "counter-current" mechanism since blood is diverted from the superficial to the deep veins, which lie in close proximity to arteries which carry the heat from the core to the periphery. In our view the present data provide no support for the theories of Wakeling and Russell (21) and Mecklenburg et al. (14) that AN produces a primary (hypothalamic) dysfunction of the temperature regulatory system. T_{re} appears to reach a plateau value independent of the initial change in H at the onset of exercise and the relative proportions of heat lost by E and $C+R$. The physiological effect of AN may be to narrow the prescriptive limits of temperature regulation, the clinical consequences

of which would be the risk of accidental hypothermia if the patient is exposed to a cool or cold environment without adequate protection.

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Influence of air flow and skin temperature on sweating at the onset, during and following exercise

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The influence of convective air flow (W_V), skin temperature ($\bar{T}_{sk}$) and rapid changes in work rate on local and whole body evaporative sweat loss ($\dot{m}_{sw}$) has been investigated in 4 healthy male subjects using the technique of resistance hygrometry.

The results showed that changes in sweat rate, measured from a capsule placed on the chest or subscapular region of the mack $\dot{m}_{sw(cap)}$, can be elicted rapidly ($t_{1/2} < 1s$) at the onset and cessation of exercise through the response is dependent on $\bar{T}_{sk}$ and the prior thermal state of the subject. During exercise, $\dot{m}_{sw}$ and $\dot{m}_{sw\,(cap)}$ were correlated and the latter variable showed a close temporal relationship with the increase in core temperature measured either in the rectum (T_{re}) or oesophagus though the association could be affected by sudden changes in W_V. At low W_V the sensitivity of $\dot{m}_{sw}$ to changes in T_{re} and $\bar{T}_{sk}$ appeared to diminish. Sudden decreases in work rate by reducing running speeds from 16 km h^{-1} to 8 km h^{-1} and 14·6 km h^{-1} to 11·3 km h^{-1} produce a transient increase in $\dot{m}_{sw\,(cap)}$ which persisted for some minutes. The rise in $\dot{m}_{sw\,(cap)}$ was associated with an increase in T_{sk} at the capsule site despite the maintenance of W_V at its high (4·5 m s^{-1}) prior level. The responses of $\dot{m}_{sw(cap)}$ and $\dot{m}_{sw}$ were little affected by directional changes in W_V, they were the same irrespective of whether the subjects ran with a following, or against a head wind. It is concluded that during exercise the integrating and modulating effects of skin temperature from different regions of the body are responsible for the control of sweat loss under conditions of constant central thermal drive. The practical applications of these results are discussed in relation to running outdoors.

1. Introduction

There have been several studies of the sweat rate response to changes in thermal state in man at rest (Belding and Hertig 1962, and Bittel and Henane 1975) and during exercise (Bullard 1962, Nielsen and Nielsen 1965, Nadel *et al.* 1971 and Saltin *et al.* 1972) using the resistance hygrometry technique which enables local sweat rate to be monitored continuously. Beaumont and Bullard (1963 and 1965) have reported that sweating is initiated within 1·5 s of the onset of muscular work, and although the magnitude of the response was influenced by mean skin temperature ($\bar{T}_{sk}$), its rate of onset was independent of the thermal load. Stolwijk and Hardy (1966) and Stolwijk *et al.* (1968) have presented a linear model of sweat control involving oesophageal temperature (T_{oes}) and $\bar{T}_{sk}$ for rest and exercise. From their model, if T_{oes} is held constant, sweat rate ($\dot{m}_{sw}$) is proportional to $\bar{T}_{sk}$ over its physiological range. The data of Belding and Hertig (1962), Bullard *et al.* (1969), Banerjee *et al.* (1969), Wurster and McCook (1969) and Nadel *et al.* (1972) support this concept, though the work of Benzinger (1969) and Wyss *et al.* (1974) casts doubts on its validity beyond a $\bar{T}_{sk}$ of 33°C. Recently Nadel *et al.* (1971) introduced a non-linear factor to their equation to

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account for the multiplicative effect of local skin cooling on central sweating drive. I find difficulty in interpreting all these data because firstly, where the experiments have been conducted at rest, $\bar{T}_{sk}$ has usually been maintained at a high level ($\sim 34°C$) in order to elicit sweating. Secondly, during exericse the work performed has been light or moderate with low air flow and with $\bar{T}_{sk}$ in excess of 30°C. This is contrary to my experience in heavier exercise with air flow sufficient to ensure evaporation of sweat. In these circumstances in a moderate environment ($T_{db} = 21°C$, rh = 50%), $\bar{T}_{sk}$ rarely exceeds 28°C (Davies 1979). Indeed, at very severe levels of exercise a cool skin appears to be a necessary physiological requirement for sustaining performance over a period of 1 h.

In the present experiments therefore I have sought to extend the work of Beaumont and Bullard (1963 and 1965) at the onset of work and examine the influence of $\bar{T}_{sk}$ during and at the cessation of moderate to severe effort in a moderate environment with high convective air flow ($2{\cdot}5$–$5{\cdot}0\,m\,s^{-1}$). In addition I also wished to examine the influences of convective air flow on local and total body sweating by observing the effects of (1) increases of air velocity at a given work load and T_{db} and (2) of running with and against a given wind speed at different T_{db} in a wind tunnel. These conditions (often in combination), are closely similar to those experienced by athletes when running outdoors, even on relatively calm days.

2. Materials and methods

Four healthy subjects were investigated (table 1) before, during and after the performance of different work loads on a motor driven treadmill. Total heat production (H) was calculated from the metabolic rate after correction for the vertical (lifting) component of the work performed. Metabolism was determined by an open circuit technique as previously described (Davies *et al.* 1976). Whole body sweating ($\dot{m}_{sw}$) was determined from weight change using a man balance. Local sweat rate ($\dot{m}_{sw\,cap}$) was determined by resistance hygrometry (Bullard 1962). A 2 cm ventilated capsule in conjunction with an infra-red water vapour pressure meter was used to record the sweat rate changes of either the chest and/or the subscapular region of the back. Different types of experiments were performed in order to establish (1) the relationship between $\dot{m}_{sw}$ and $\dot{m}_{sw\,(cap)}$ over 1 h. These experiments were performed on all four subjects. Local sweat rate was recorded from the chest, and body weight changes were determined at 30 and 60 min of exercise. Core temperature was measured from a thermocouple placed either in the oesophagus (T_{oes}) or rectum (T_{re}) and the mean skin temperature ($\bar{T}_{sk}$) was determined from measurements taken at 13 sites. In these experiments the T_{oes} or T_{re} and $\bar{T}_{sk}$ at sweat onset was measured. The effect of artificial acclimatization to heat on $\dot{m}_{sw}/\dot{m}_{sw\,(cap)}$ was also noted in subject 3. (2) The

Table 1. Physical characteristics. Age, weight (wt), height (ht) and maximal oxygen intake ($\dot{V}O_2$ max—$l\,min^{-1}$) and aerobic power output (W).

Subject	Age (yr)	Wt (kg)	Ht (cm)	$\dot{V}O_2$ max $l\,min^{-1}$	W
1	32	74	171	4·00	1364
2	22	67	180	3·80	1296
3	44	62	178	3·90	1330
4	32	65	173	4·72	1610

time course of changes in $\dot{m}_{sw\,(cap)}$ at the onset of muscular work, and the influence on this of local skin temperature measured at the capsule site $T_{sk\,(cap)}$ and $\bar{T}_{sk}$ was examined on two subjects (3 and 4). Sweating was induced by light exercise and then the effects of sudden increases in work load were observed. For these experiments the capsule was placed on the subscapular region of the back. Abrupt changes in $T_{sk\,(cap)}$ and $\bar{T}_{sk}$ were achieved by rapid increases in convective air flow from a fan placed immediately above the subject. The time course of the fall in $\dot{m}_{sw\,(cap)}$ during recovery from exercise to a baseline either of rest or light exercise at different levels of air flow was observed, and (3) the effect of different levels of convective air flow at a given work load sustained for 1 h and the directional influence of air flow were investigated on the same two subjects. In these experiments the subjects were required to run with and against a given wind speed on a treadmill placed inside a wind tunnel; local sweating was monitored from two capsules, one placed on the chest and the other on the subscapular region of the back.

3. Results

The relationship between whole body and local (chest) sweating is shown in figure 1. For the 4 subjects measured the association between the two variables is strong, the correlation coefficient (r) varies from 0·61–0·99 but the interindividual differences in the slope of the regressions are large (range 0·00018–0·00037). This reflects the difference in sensitivity of the chest site to sweating in the four subjects. The most sensitive subject was an athlete in regular training and the least sensitive was 12–22 yr older than the other men in the investigation. Following the period of artificial acclimatization the latter subject's local (chest) sweat production was improved, but it should be noted

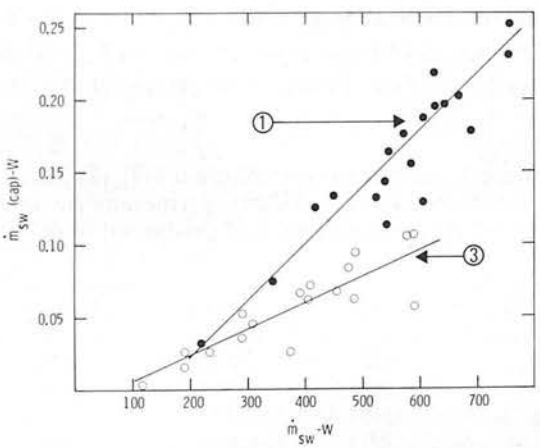

Figure 1. The relationship between local sweat loss measured under a ventilated capsule placed on the pectoral region of chest ($\dot{m}_{sw(cap)}$) and whole body sweat loss ($\dot{m}_{sw}$) determined from clothed weight changes before and after a 1 h period of exercise at a constant rate. Subject 3 was measured before and after a period of acclimatization. The observed points for subject equations for the 4 subjects studied were: (1) $\dot{m}_{sw(cap)}$ (W) $= -0{\cdot}252 + 0{\cdot}00037\ \dot{m}_{sw}$ (W); r = 0·92. (2) $\dot{m}_{sw(cap)}$ (W) $= -0{\cdot}003 + 0{\cdot}00024\ \dot{m}_{sw}$ (W); r = 0·99. (3) $\dot{m}_{sw(cap)}$ (W) $= -0{\cdot}012 + 0{\cdot}00018\ \dot{m}_{sw}$ (W); r = 0·86 (before acclimatization), $\dot{m}_{sw(cap)}$ (W) $= -0{\cdot}007 + 0{\cdot}00022\ \dot{m}_{sw}$ (W); r = 0·78 (after acclimatization). (4) $\dot{m}_{sw(cap)}$ (W) $= -0{\cdot}057 + 0{\cdot}00028\ \dot{m}_{sw}$ (W); r = 0·61.

(figure 1) that even after this procedure his $\dot{m}_{sw\,(cap)}/\dot{m}_{sw\,(body)}$ relationship remained to the right of subject 1 who was active, but not in training. However, it should also be emphasized that the degree of sensitivity of the chest site to sweating was without effect on their whole body sweat ($\dot{m}_{sw}$)/total body heat production (H) relationship. For the 4 subjects measured under the same environmental conditions ($T_{db}=21°C$, $T_{wb}=15°C$, wind velocity $=2{\cdot}5-5{\cdot}0\,m\,s^{-1}$) the $\dot{m}_{sw}/H$ regression lines were not significantly different ($p>0{\cdot}5$) and they could be combined.

3.1. *Time to sweat onset*

The time to sweat onset was related to the severity of exercise under given environmental conditions and varied from 3·7 to 15·7 min. Equally, for a given exercise, the pattern of variability of time to sweat onset was the same as the above when the severity of the environmental conditions was increased. However, for a given subject the core temperature (T_{re} or T_{oes}) at sweat onset was constant and independent of exercise intensity and environmental conditions over the range studied (table 2). Moreover, if sweating was induced prior to exercise (by heat or work) $\dot{m}_{sw\,(cap)}$ responded to an increase in work load within 2 s. The response could be modified (but not inhibited) by a sudden and simultaneous decrease in $T_{sk\,(cap)}$ at the onset of exercise (figure 2).

3.2. *Exercise sweating*

During exercise $\dot{m}_{sw(cap)}$ invariably reached a steady state within 40 min and showed a close temporal relationship to T_{re} and T_{oes}. A sudden reduction in local and mean skin temperatures produced by a rapid change in convective air flow resulted in a marked and instantaneous decrease in $\dot{m}_{sw\,(cap)}$ (figure 3) but the effect was greater at the lower than at the higher work loads. The decrease in $\bar{T}_{sk}$ had little effect on core temperature at high exercise levels but there was a small but systematic increase ($\sim 0{\cdot}2°C$) in T_{oes} during lighter work. This was in contrast to the changes in T_{re} and $\dot{m}_{sw}$ produced by different levels of convective air flow during 1 h of exercise (table 3).

Table 2. Rectal temperature (T_{re}) and mean skin temperature ($\bar{T}_{sk}$) and the time to sweat onset (t_{so}) in relation to (A) different total heat production in the same environmental conditions ($T_{tb}=21°C$, rh 50%) and (B) the same total heat production in differing environmental conditions. Subject 3.

	T_{db} (°C)	H (W)	T_{re} (°C)	$\bar{T}_{sk}$ (°C)	t_{so} (min)
A	21	488	37·25	29·81	15·7
		492	37·03	29·36	12·0
		589	37·15	28·77	9·0
		791	36·89	29·47	5·8
		883	37·05	28·88	6·1
		983	37·00	28·54	5·3
B	20	1002	37·10	27·39	13·0
		1000	37·00	27·35	13·5
	25	1001	37·18	28·48	7·0
		1002	37·40	30·35	7·5
	30	984	37·25	31·37	3·7
		1004	37·20	30·84	3·3

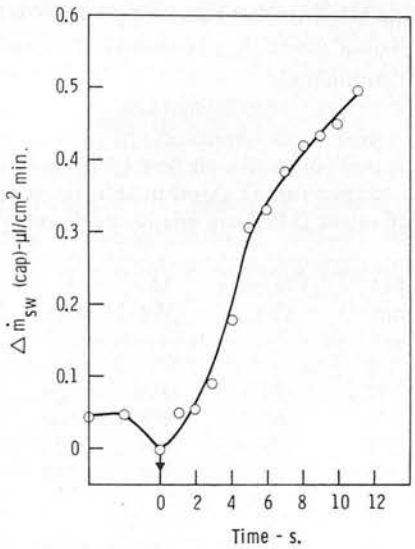

Figure 2. Change of sweat rate, $\Delta\ \dot{m}_{sw\ (cap)}$ measured from a ventilated capsule placed on the subscapular region of the back, after a sudden change of work rate (indicated by the arrow) from running at 11·3 km h^{-1} to 14·6 km h^{-1} on the level.

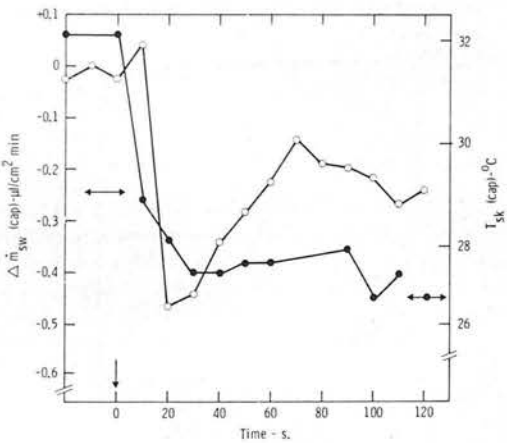

Figure 3. Effect of suddenly changing local skin temperature $T_{sk\ (cap)}$ (indicated by ↓ on the abscissa) during exercise at 12·2 km h^{-1} on local (subscapular sweat rate ($\dot{m}_{sw\ (cap)}$). Symbols (●) $T_{sk\ (cap)}$; (○) $\dot{m}_{sw\ (cap)}$. Arrows (⟷) indicate mean skin temperature ($\bar{T}_{sk}$) levels.

Increased convective air flow reduced $\bar{T}_{sk}$ and T_{re} but $\dot{m}_{sw}$ calculated from clothed weight loss (see methods) remained unchanged. Thus, the sensitivity of the whole body sweating mechanism changed in the sense that at high air flows a smaller rise in T_{re} and $\bar{T}_{sk}$ was sufficient to produce a given $\dot{m}_{sw}$ necessary for heat balance than observed when air movement was diminished.

Table 3. The effects of increased convective air flow (W_v) on metabolic (M) and total heat production (H), rectal temperature (T_{re}), mean skin temperature ($\bar{T}_{sk}$), and sweat loss ($\dot{m}_{sw}$). Values for oxygen intake ($\dot{V}O_2$) and relative work load ($\%\dot{V}O_2$ max) are also given.

Subject	W_v (m/s)	$\dot{V}O_2$ (l min^{-1})	$\%\dot{V}O_2$ max (%)	M (W)	H (W)	T_{re} (°C)	$\bar{T}_{sk}$ (°C)	$\dot{m}_{sw}$ (W/m^2)
3								
(T_{db}=25°C	1·0	2·76	71	943	925	39·11	32·14	295
T_{wb}=17°C)	3·0	2·70	69	919	900	38·90	29·48	330
	4·0	2·78	71	948	928	38·88	29·32	293
	7·0	2·73	70	930	912	38·41	28·87	309
4								
(T_{db}=21°C	1·0	3·31	70	1129	1129	39·31	29·73	399
T_{wb}=15°C)	2·0	—	—	—	—	39·04	29·05	374
	4·5	3·18	67	1084	1084	38·65	27·32	398
	7·0	3·12	66	1063	1063	38·76	26·93	413

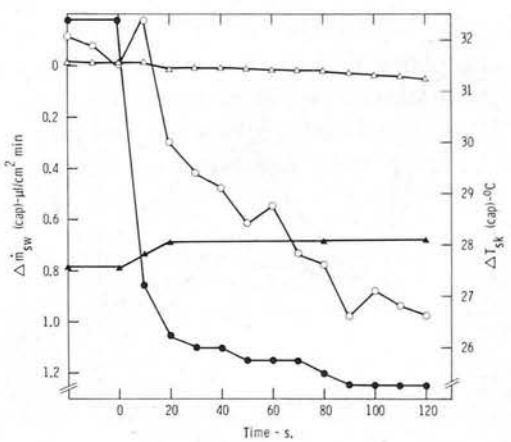

Figure 4. Change in $\dot{m}_{sw\ (cap)}$ and local skin temperature $T_{sk\ (cap)}$ during recovery from exercise to a baseline of rest in the absence of convective air flow (△) $\dot{m}_{sw\ (cap)}$, (▲) $T_{sk\ (cap)}$ and with convective air flow maintained at a rate equivalent to the running speed during the exercise (○) $\dot{m}_{sw\ (cap)}$, (●) $T_{sk\ (cap)}$.

3.3. *Recovery sweating*

At the cessation of exercise, recovery $\dot{m}_{sw\ (cap)}$ was maintained for a period of at least 1–2 min at the level observed during the steady state of work. The maintenance of local sweat rate during this period appeared to be associated with a slow decline in T_{oes} and T_{re} and a rapid increase in $T_{sk\ (cap)}$ (figure 4). These observations were made under

conditions (as in outdoor running) where the air flow was immediately reduced on cessation of effort to near zero levels. If air flow was maintained during the recovery period, the sweat rate decreased rapidly, the rate of decline being associated with the change in $T_{sk\ (cap)}$ (figure 4). In contrast, if recovery from hard exercise was performed to a baseline of light exercise (16 km h^{-1} at 0% on the treadmill) instead of rest and air flow was maintained (4·5 ms^{-1}), $\dot{m}_{sw\ (cap)}$ invariably rose (figure 5). However, again this change in $\dot{m}_{sw}$ was associated with a rise in $T_{sk\ (cap)}$ despite the maintenance of the prior high level of convective air flow.

Finally, in order to investigate the effects of running for 1 h with and against convective air flow, as is often experienced in outdoor running, experiments were performed on a reversible treadmill housed within a wind tunnel capable of producing

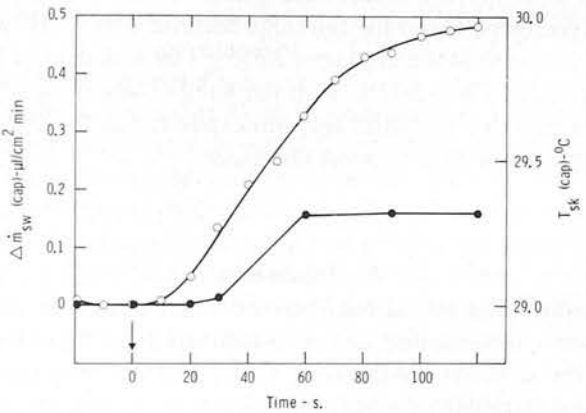

Figure 5. The increases in (○) $\dot{m}_{sw\ (cap)}$ and (●) $T_{sk\ (cap)}$ after reducing the work load by decreasing speed from 14·6 to 11·3 km h^{-1}. The arrow marks the change of work load.

Table 4. Comparison of the metabolic and thermal responses to exercise (A) running against and (B) with a convective air flow at a velocity equivalent to the subject's speed on the treadmill. Symbols as for table 3, with the addition of local sweat loss from the pectoral (capsule) region of the body $\dot{m}_{sw\ (cap)}$. The experiments were performed at 12·9 km h^{-1} (subject 3) and 14·5 km h^{-1} (subject 4) at 0% gradient, thus M was equivalent to H and has been omitted from the table. n indicates the number of experiments.

Subject		$\dot{V}O_2$ (l min^{-1})	$\%\dot{V}O_2$ max (%)	H (W)	T_{re} (°C)	$\bar{T}_{sk}$ (°C)	$\dot{m}_{sw}$ (W)	$\dot{m}_{sw\ (cap)}$ (W)
3	A (n=6)	2·97 ±0·07	76 ±2	1022 ±35	38·81 ±0·11	29·13 ±1·65	553 ±155	0·14 ±0·06
	B (n=6)	2·88 ±0·08	74 ±2	984 ±27	38·77 ±0·18	29·39 ±1·34	571 ±185	0·13 ±0·03
4	A (n=6)	3·38 ±0·10	72 ±2	1154 ±37	38·75 ±0·31	28·05 ±2·06	686 ±138	0·27 ±0·10
	B (n=6)	3·12 ±0·08	69 ±3	1066 ±28	38·60 ±0·30	28·31 ±2·34	623 ±154	0·25 ±0·09

unidirectional (approximately) laminar air flow under different environmental (T_{db} and T_{wb}) conditions. $\dot{m}_{sw\,(cap)}$ was monitored from the pectoral and subscapular regions of the body. The results are summarized in table 4. In both subjects M was higher running against than with the wind. The effect is greater in subject 4 than 3 who exercised at a higher speed (14·4 km h^{-1}) and therefore (since air flow was maintained at the velocity of the runner) performed more work against air resistance. If this factor is taken into account both subjects showed the same T_{re}, T_{sk}, $\dot{m}_{sw}$, and $\dot{m}_{sw\,(cap)}$ (pectoral) for the two conditions. However, both subjects sweated more on the front of their bodies than the back whether they ran with or against the wind: $\dot{m}_{sw}$ (pectoral W) = 0·059 + 0·859 $\dot{m}_{sw}$ (subscapular W) $r = +0{\cdot}71$.

This phenomenon was associated with a higher T_{sk} at the capsule site. Thus, subject 3 running against a head wind had a pectoral T_{sk} of 30·16 ± 1·64°C and a subscapular T_{sk} of 29·33 ± 0·76°C which were similar to the values (30·31 ± 1·61°C and 28·75 ± 1·13°C respectively) found for the same exercise performed with a following wind. The respective figures in subject 4 were 28·55 ± 1·99°C and 26·91 ± 2·51°C against and 29·09 ± 1·83°C and 27·00 ± 2·52°C with the wind. The core temperature (T_{re}) at sweat onset for the two sites (pectoral and subscapular) was the same irrespective of whether the subjects ran with or against the wind.

4. Discussion

The results confirm and extend the observations of Beaumont and Bullard (1963 and 1965). They show that sweating can be elicted rapidly (within 2·0 s) at the onset of exercise, though the response is dependent on $\bar{T}_{sk}$ and the prior thermal state of the subject. If exercise is started from a condition of rest in a moderate environment, sweat onset is delayed by 5 to 15 min and is dependent on the rate of work (table 2). During exercise $\dot{m}_{sw\,(cap)}$ shows a close temporal relationship to the increase in T_{re} and T_{oes} though the association between the two variables can be influenced by transient changes in convective air flow and $T_{sk\,(cap)}$ (figure 3). However, provided the change in convective air flow (W_v) is sustained for a (1 h) period of time the final whole body sweat loss ($\dot{m}_{sw}$) seems to be remarkably unaffected (table 3). At reduced and enhanced convective air flows, for a given H, $\dot{m}_{sw}$ appears to reach a similar final value. The difference in the two conditions is the level of central and peripheral drive necessary to produce $\dot{m}_{sw}$. At low air flows T_{re} and $\bar{T}_{sk}$ are 0·75°C and 3·3°C approximately and respectively higher than when performing the same work under conditions of greater air movement (table 3). These results suggest in agreement with Nadel and Stolwijk (1973) that the sweat mechanism becomes less sensitive as air flow is diminished though surprisingly the responses of $\dot{m}_{sw}$ and $\dot{m}_{sw\,(cap)}$ are little affected by the directional influence of W_v (table 4). The local sweat loss from the front of the body and $\dot{m}_{sw}$ were the same (if account was taken of the small differences in M) irrespective of whether the subjects ran with or against a given W_v. $\dot{m}_{sw}$ retained its relationship to H in the two conditions, though in absolute terms the $\dot{m}_{sw\,(cap)}$ was always greater from the pectoral than the subscapular region. This could be associated with the different local T_{sk} at the two sites.

At the cessation of work to a baseline of rest, the decline in $\dot{m}_{sw\,(cap)}$ was delayed in the absence of convective air flow (as is the case in outdoor running) but if W_v was maintained at a rate equivalent to the prior exercise running speed, the decrease in $\dot{m}_{sw\,(cap)}$ was rapid and closely associated with the fall in $T_{sk\,(cap)}$ at the capsule site

(figure 4). In contrast, if the subject was allowed to recover to a baseline of lighter exercise and with W_v maintained $\dot{m}_{sw\,(cap)}$ actually increased (figure 5). This rise in $\dot{m}_{sw\,(cap)}$ persisted and did not decline to the expected level appropriate for the reduced work output during the time of observation. However, again this change in local sweat response was associated with the rise in T_{sk} at the capsule site and $\bar{T}_{sk}$.

These results underline the critical influence of both local T_{sk} and $\bar{T}_{sk}$ on $\dot{m}_{sw(cap)}$ and $\dot{m}_{sw}$ during exercise and support the view (Nadel *et al.* 1971) that the integration of skin temperature from different regions of the body is responsible for the final modulation of sweat loss against a background of constant central thermal drive during work at a given rate. The sweat output from a given skin area is closely dependent on the local skin temperature. During heavy exercise in a moderate environment, the local skin temperature in different areas of the body can vary over wide limits and the sensitivity of a given site will vary within and between individuals. This can be seen from figure 1 and yet for the experiments reported in this study $\dot{m}_{sw}$ was always closely associated with H. The central (T_{re} or T_{oes}) temperature drive was the same in all subjects if account was taken of the relative work load. These observations can only be reconciled if one assumes that the $\dot{m}_{sw}$ is effected by the rise in core temperature but the actual regional losses are determined by local blood flow and temperature. Seen in this light during moderate exercise thermal regulation is essentially a balance between local and central drive. The metabolic heat production and rate of blood flow determine the thermal gradient between the core and skin. During light exercise where blood flow to active muscles is not a limiting factor the flow of heat from the core to the periphery raises $\bar{T}_{sk}$, stimulates sweating and ultimately a balance is reached whereby the heat delivered to and removed from the skin are equal. However, during heavy exercise where a cool skin is prerequisite for sustained performance (Davies 1979), temperature regulation will be governed not only by central stimulation by peripheral inhibition of the evaporative and vasomotor responses. Thermal balance will be a compromise between the body's heat dissipating and conservation mechanisms. The nature of the compromise is probably determined by the requirements of the working muscles for blood flow. Under these conditions a thermal gradient will be established as for light exercise, but peripheral vasoconstriction will increase the core–skin temperature difference and reduce heat dissipation by local sweat suppression. To overcome the increased conservation of heat by skin cooling, the core temperature rises to increase heat dissipation and restore thermal equilibrium.

5. Practical implications

The present experiments were designed, in part, to study sweating under conditions encountered by athletes when running out of doors, hence the observations on the directional effect of air flow (table 4), the immediate onset and cessation of effort (figures 2 and 4) and changes in pace (figure 5). It would seem from the results that the unidirectional effect of air flow (as encountered outdoors when running on a calm day) has little or no effect on the whole body or total sweat response. The pattern of sweating in the present subjects was the same whether they ran with or against the wind (table 4). The rate of sweat onset was determined by the prior thermal state of the subject, its cessation at the end of exercise was dependent on the convective air flow which suggests that from a thermoregulatory point of view, the short periods of warming up practised by the majority of athletes is physiologically sound. Finally, slowing down (changing

pace) during a race will undoubtedly ease the metabolic load on the subject, but it is not without thermal consequences and may paradoxically result, at least in the short term, in an increase in the thermal input to the sweat regulatory system.

The author is indebted to the subjects for their co-operation and at various times to E. ZeidiFard, M. Thompson, M. Gibbons and M. White for their assistance with the experiments.

L'influence du flux d'air par convection (W_v), de la température cutanée ($\bar{T}_{sk}$) et des variations rapides dans la cadence de travail sur la perte évaporatoire par sudation ($\dot{m}_{sw}$) soit locale ou totale, a été étudiée sur quatre sujets masculins, à l'aide de la technique par hygrométrie à résistance.

Les résultats ont montré que les variations dans la vitesse évaporatoire, mesurée au moyen d'une capsule fixée sur la poitrine ou dans la région sous-scapulaire du dos $\dot{m}_{sw(cap)}$, peuvent être provoquées rapidement ($t_{1/2} < 1$ s) au début et à la fin d'un exercice, bien que la réponse dépende de $\bar{T}_{sk}$ et de l'état thermique antérieur du sujet. Pendant l'exercice, $\dot{m}_{sw}$ et $\dot{m}_{sw(cap)}$ sont correlés et la dernière variable présente une relation étroite avec les accroissements de la température du noyau évaluée à partir de la température rectale ou oesophagienne, bien que cette association puisse être affectée par des variations brusques de W_v. Pour des W_v faibles, la sensibilité de $\dot{m}_{sw}$ vis-à-vis des variations de la T_{re} et la T_{sk}, semble diminuer.

Des diminutions subites du travail en réduisant les vitesses de course de 16 km h^{-1} à 8 km h^{-1} et de 14,6 km h^{-1} à 11,3 km h^{-1}, entraînent un accroissement transitoire de $\dot{m}_{sw(cap)}$ qui persiste pendant quelques minutes. L'accroissement de $\dot{m}_{sw(cap)}$ était associé avec un accroissement dans $\bar{T}_{sk}$ dans la zone de la capsule, en dépit du maintien de W_v à son niveau haut (4,5 m s^{-1}) antérieur. Les réponses de $\dot{m}_{sw(cap)}$ et de $\dot{m}_{sw}$ ont été peu affectées par les variations de direction de W_v que les sujets courussent suivis par un autre ou qu'ils eussent le vent en face, ces réponses étaient les mêmes. On conclut que pendant l'exercice, les effects intégratifs et modulants de la température cutanée dans différents zones du corps sont responsables de la commande de la perte évaporatoire par sudation sous la condition d'une température centrale constante. La discussion porte sur l'applicabilité de ces résultats à des situations de course en plein air.

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Effects of wind assistance and resistance on the forward motion of a runner

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DAVIES, C. T. M. *Effects of wind assistance and resistance on the forward motion of a runner.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 48(4): 702–709, 1980.—The aerobic energy cost ($\Delta\dot{V}O_2$) of running at different speeds (V) with and against a range of wind velocities ($W_{\dot{V}}$) has been studied in a wind tunnel on three healthy male subjects and the results compared with downhill and uphill gradient running on a motor-driven treadmill. In terms of equivalent horizontal and vertical forces, comparison showed that the two forms of exercise were physiologically identical for gradients and W_V ranging from −10 to +5% and 1.5 to 15 $m \cdot s^{-1}$, respectively. The apparent mechanical efficiencies of the work performed with a head and following wind were approximately +0.35 and −1.2. At $W_V >$ 15 $m \cdot s^{-1}$ it was more efficient to run against the wind and the corresponding gradient on the treadmill. At high W_V the subjects altered their posture and "leaned" into the wind, thus possibly converting potential drag into body lift. The energy cost of overcoming air resistance on a calm day outdoor was calculated to be 7.8% for sprinting (10 $m \cdot s^{-1}$), 4% middle-distance (6 $m \cdot s^{-1}$), and 2% marathon (5 $m \cdot s^{-1}$) running.

oxygen intake; aerobic exercise; gradient; treadmill; positive and negative work

THE AEROBIC ENERGY COST of level and gradient exercise has been studied many times (see Ref. 12), but the influence of wind resistance on oxygen intake ($\dot{V}O_2$) has received comparatively little attention. Margaria (9) discusses the problem on the basis of Fenn's early observations (6, 7) and Hill's (8) work with a model of a runner in a wind tunnel, in which it was suggested that there might be an exact equivalence between working against horizontal forces produced by different air velocities (W_V) outdoor and the vertical forces involved in gradient exercise on a conventional (indoor) laboratory treadmill where air resistance is effectively eliminated, but only Pugh (13, 14), to the author's knowledge, has made observations of this type. Pugh (13, 14) found in four subjects exercising on a treadmill housed inside a wind tunnel that the energy cost of a head wind was proportional to W_V^2, but the effect was dependent on the runners' speed (V), and no precise equivalence between gradient and level "wind-resistance" running could be seen. The horizontal work against the wind was always performed more efficiently than the vertical work against gravity. Further, Pugh (14) calculated that the energy cost of overcoming air resistance outdoors and suggested, even at middle-distance speeds, that it might account for at least 8% of the total $\dot{V}O_2$, a value four times in excess of Hill's (8) original prediction. It is therefore of interest that recently McKiken and Daniels (11) have failed to show any differences between measured $\dot{V}O_2$ for outdoor track and indoor treadmill running over a range of long- and middle-distance speeds. Further no one to the author's knowledge has considered or attempted to measure the effects of a following wind on the forward motion of a runner.

To gain further information on the effects of a head and following wind on the forward motion of a runner, the present investigation was planned in three parts. *1)* Experiments were conducted on three healthy male subjects who ran on a motor-driven treadmill with and against wind velocities equivalent to their running speed; this type of running is often encountered outdoor. *2)* The experiments were extended to very high (gale-force) head and following winds similar in intensity to those experienced by climbers. *3)* Comparison of the work with and against the horizontal forces encountered in *1* and *2* were made with the vertical work of running with and against gravity during downhill and uphill gradient exercise, respectively, on the same subjects.

MATERIALS AND METHODS

The physical characteristics of the three healthy male subjects studied are given in Table 1. *Subjects 1* and *2* were endurance athletes in regular training (100–150 miles/wk) and *subject 3* was active and ran regularly for pleasure. Measurements were normally made after a light meal. The experiments were conducted over a 12-mo period at two laboratories. The level and gradient running experiments were carried out at the author's laboratory and the wind resistance studied at the Institute of Aviation Medicine, Farnborough. In the level and gradient running experiments, the subjects ran continuously at set speeds and the slope of the treadmill bed was raised every 10 min by approximately 2% increments. Both negative (downhill) and positive (uphill) gradients were investigated at zero wind resistance and $\dot{V}O_2$ levels were measured during the final 4 min at each load using the standard (Douglas bag) open-circuit technique. Duplicate (and more often triplicate) Douglas bag samples were taken at each load and the data presented are the mean $\dot{V}O_2$ values for each collection period. The experiments running with and against a wind were made in a

BLE 1. *Physical characteristics of subjects*

bj No.	Wt, kg	Ht, cm	A_D, m^2	$\dot{V}O_{2\,max}$, l·min^{-1}
1	65	173	1.78	4.72
2	66	185	1.88	5.01
3	62	178	1.78	3.90

A_D, DuBois surface area; $\dot{V}O_{2\,max}$, maximal aerobic power output.

matic chamber where the airstream produced by a gh-power (propeller) fan was deflected through an an- of 90° by a set of vertical vanes 3 m in front of the admill. Wind velocities of 1.5–18.5 m·s^{-1} (40 mph) re available. The airflow was turbulent particularly at gh velocities; vane anemometer readings fluctuated by m·s^{-1} across the chamber at the highest fan setting. he subjects again ran continuously and the same pro- col (including the methods for measuring $\dot{V}O_2$) as de- ribed for gradient running was used. The fan setting nd therefore airflow) was increased every 10 min and e subject maintained a constant pace on the treadmill zero grade. Usually five fan settings and two running readmill belt) speeds were studied (on different days) d the experiments were repeated with the mill of the admill reversed. The same fan settings were used for ch set of experiments and air velocity was checked veral times at each setting at representative points at e head of the treadmill. The projected area of the bjects running with and against the wind was estimated om photographs taken during the experiments beside rectangular surface of known area following the method Pugh (13).

Calculations from the raw data were made on the llowing basis. Air resistance or drag (D) is proportion αW_V^2, where W_V is the wind velocity and α is the roportionality constant for a given object. The relation- hip between D and W_V is normally expressed in terms the drag coefficient (C_D); thus $C_D = D/\beta A_r$, where β is he dynamic pressure and is equal to $0.5\rho W_V^2$ and A_r is he projected area of the runner. ρ is the air density. hus, if D is expressed in kilogram force and A_r in square eters, then

$$C_D = \frac{\alpha}{0.5\rho\ A_r} = \frac{\alpha}{0.0625\ A_r} \qquad (1)$$

n this study α was obtained by constructing graphs elating $\dot{V}O_2$ to W_V^2 and $\dot{V}O_2$ to (lifting) work rate ($\dot{W}$) on he treadmill. Values of W_V^2 and $\dot{W}$ at equal $\dot{V}O_2$ were ead off and $\dot{W}$ was divided by V to obtain the total force F) opposing motion. α was calculated at the slope of the F/W_V^2 line. The confounding influence of a change in Reynolds' number (R) on D was avoided in the present xperiments by confining the W_V range from 1.5 to 18 m· -1. From laboratory experiments on cylindrical models imilar in overall dimensions to man, the effects of R on C_D are relatively constant: critical Reynolds' numbers re only reached at W_V in excess of 18.5 m·s^{-1}. The nterested reader is referred to Pugh (14) for a more detailed treatment and analysis of the problem of calcu- ating D and the influence of R during treadmill running n a wind tunnel.

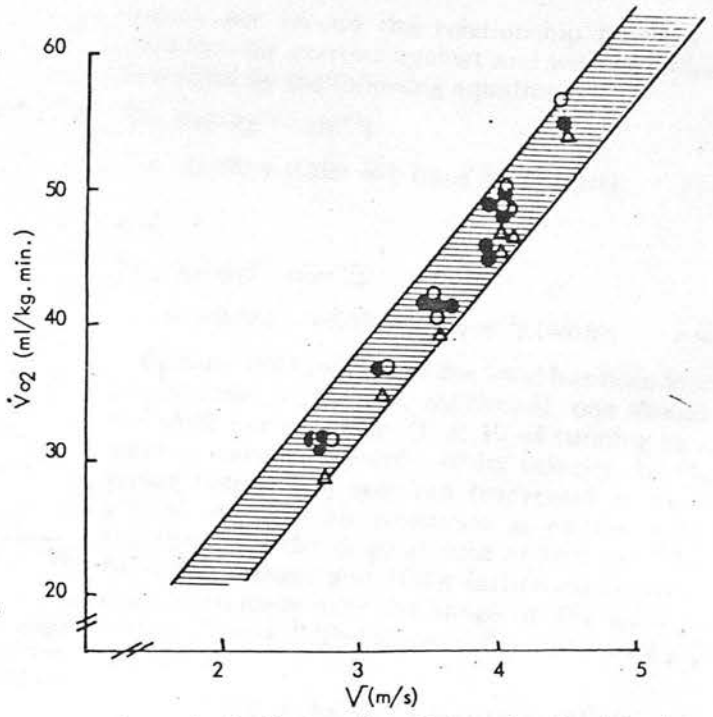

FIG. 1. Oxygen intake ($\dot{V}O_2$) running with (△) and against (○) a given wind velocity equivalent to speed of Farnborough treadmill. Unit treadmill (●) at minimal air velocity (see METHODS). *Hatched area* represents intrasubject variation (95% confidence limits) of $\dot{V}O_2$ against a speed for subject measured on 33 separate occasions on Unit treadmill. Comparison of two treadmills used in this investigation at minimal airflows for each subject over a range of speeds yielded no statistically significant differences.

RESULTS

The effects on $\dot{V}O_2$ of running with and against a wind at a velocity equivalent to the speed of the treadmill (i.e., below 5 m·s^{-1}) were marginal and certainly within the intrasubject variation of $\dot{V}O_2$ for running at minimal W_V on the two treadmills used in this investigation (Fig. 1). However, as W_V increased the cost of work, the $\dot{V}O_2$ rose as a curvilinear function of W_V (Fig. 2). At high air velocities (>15 m·s^{-1}) $\dot{V}O_2$ tended to plateau and approach an asymptotic value. The effect of increasing the treadmill speed (V) was a parallel displacement of the $\dot{V}O_2/W_V$ curve to the left. Thus, the effects of V could be removed by plotting the change in oxygen intake ($\Delta\dot{V}O_2$) from a base line of minimal W_V for each exercise intensity. Running with the wind effected a curvilinear reduction in $\dot{V}O_2$, but the changes were less marked than for exercise against a head wind. Changes in V produced an upward displacement in the $\dot{V}O_2/W_V$ curve.

The curvilinear nature of the $\dot{V}O_2/W_V$ curve (but not the plateau effect) could be removed by considering $\Delta\dot{V}O_2$ (ml·kg^{-1}·min^{-1}) as a function of W_V^2 at the different levels of V used in this study. The relationship between the two variables for running with and against the wind are shown in Fig. 3. $\Delta\dot{V}O_2$ is essentially a linear function of W_V^2 over the range of W_V from 5 to 15 m·s^{-1}; at the higher W_V the increase in $\Delta\dot{V}O_2$ diminishes and $\Delta\dot{V}O_2/W_V^2$ relationship levels off. The association of $\Delta\dot{V}O_2$ with W_V^2 is independent of V and if $\Delta\dot{V}O_2$ is expressed in milliliters per kilogram (body wt) per minute and W_V^2 in

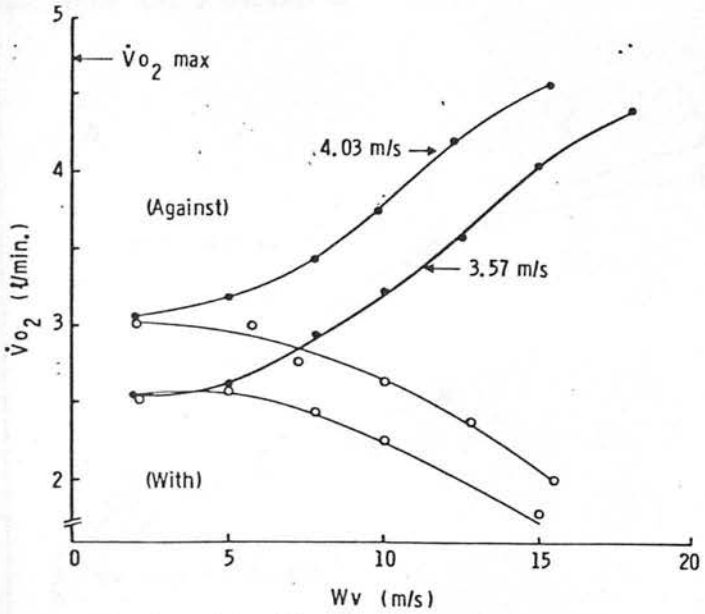

FIG. 2. Typical relationship of $\dot{V}O_2$ to wind velocity (W_V) over range studied in this investigation (*subj 1*). Data points are shown for two treadmill speeds (3.57 and 4.03 $m \cdot s^{-1}$), with (○) and against (●) the wind. *Arrow* marks $\dot{V}O_{2\,max}$ of subject.

meters per second the relationship between the two variables for exercise against and with the wind can be described by the following equations

$$\dot{V}O_2\ (ml \cdot kg^{-1} \cdot min^{-1}) = -0.700 + 0.109\ W_V^2\ (m \cdot s^{-1})\ \text{(against)}; \qquad r = +0.993$$

and

$$\dot{V}O_2\ (ml \cdot kg^{-1} \cdot min^{-1}) = -0.054 - 0.655\ W_V^2\ (m \cdot s^{-1})\ \text{(with)}; \qquad r = -0.954$$

Because the force (F) of the wind has been found to be proportional to W_V^2 (see METHODS), one should expect the work per unit time ($F \times V$) of running against the wind to vary as the cube of its velocity. In Fig. 4 the power output ($\dot{W}$) required (expressed as $kg \cdot m \cdot kg^{-1} \cdot s^{-1}$) to overtake air resistance is plotted against W_V^3 together with the original data of Hill (8). The results from the present and Hill's earlier experiments are in close agreement over the range of W_V up to approximately 12 $m \cdot s^{-1}$. The slope of the curve over this range is given by

$$\dot{W}\ (kg \cdot m \cdot kg^{-1} \cdot s^{-1}) = 0.00034\ W_V^3\ (m \cdot s^{-1})$$

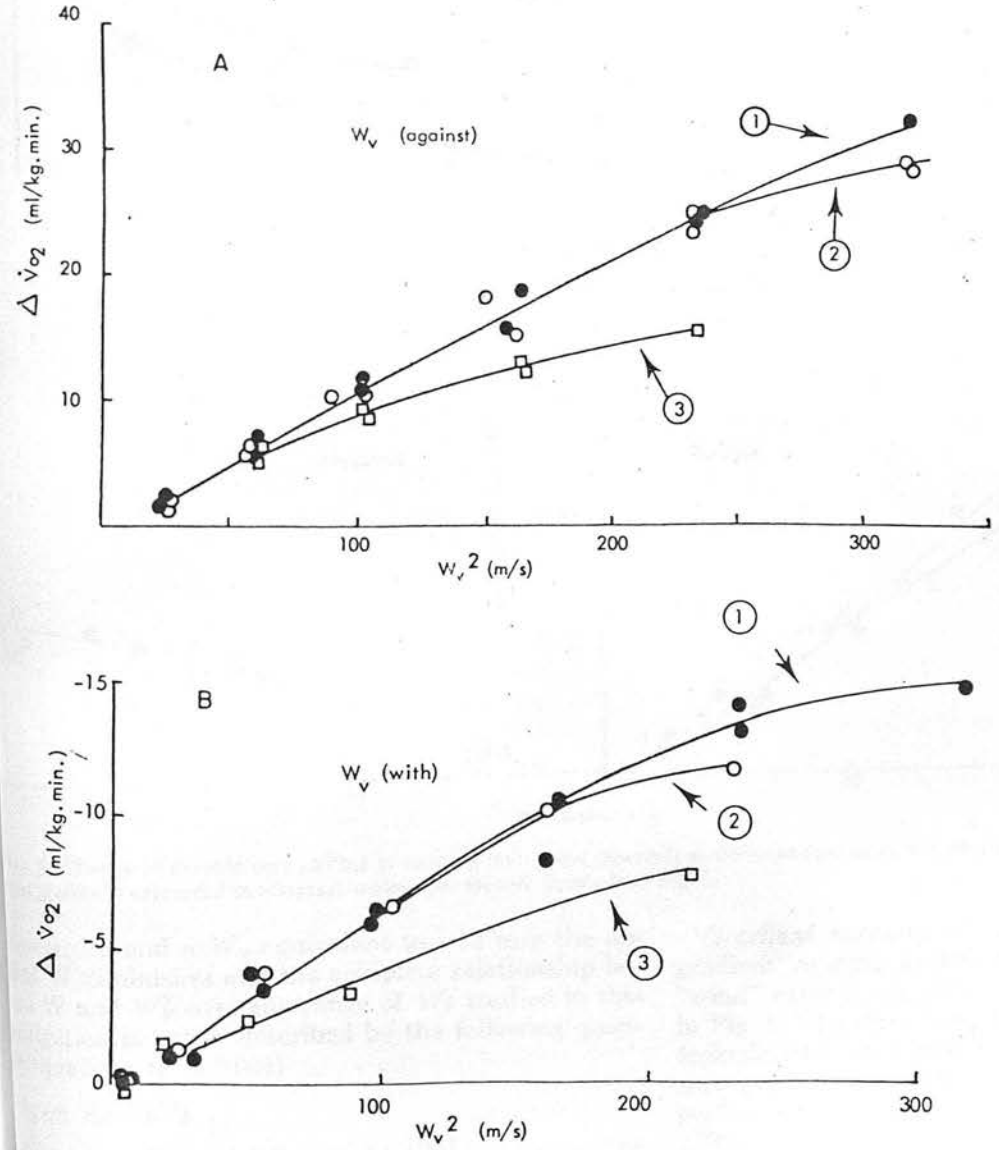

FIG. 3. Change in oxygen intake ($\Delta\dot{V}O_2$) in relation to W_V^2 for *subj 1* (●), *subj 2* (○), and *subj 3* (□) against (*A*) and with (*B*) the wind.

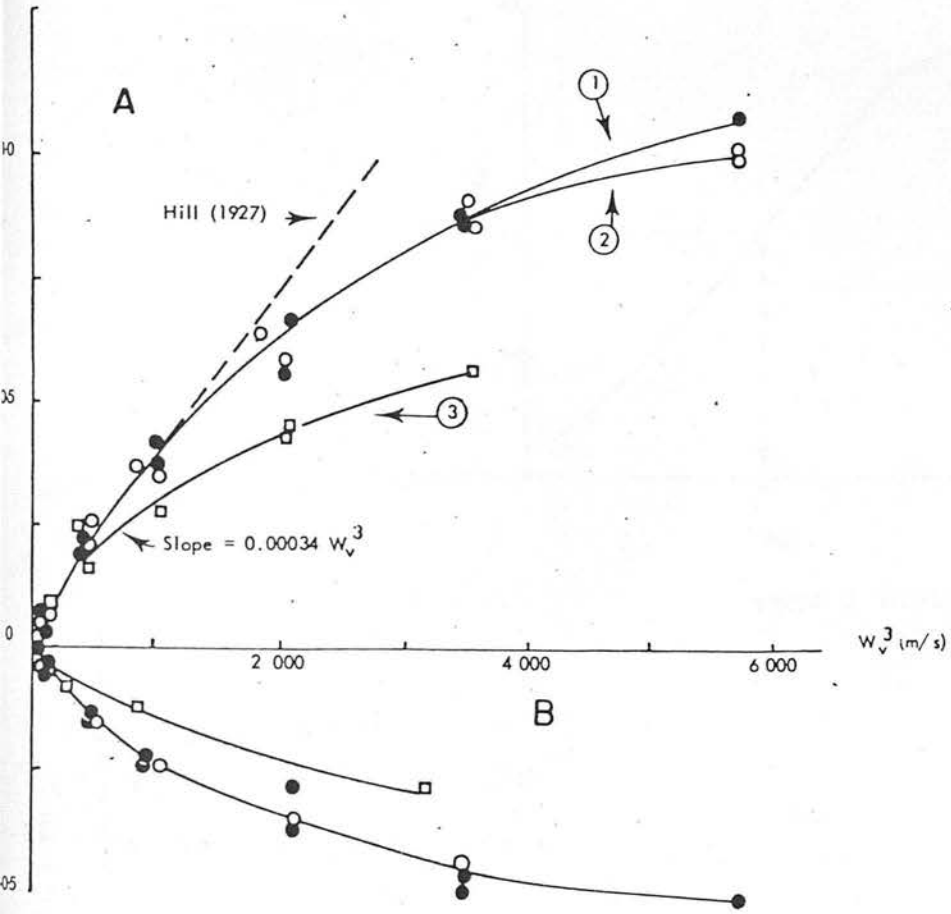

FIG. 4. Work required either to overcome (*A*) or to resist (*B*) air velocity. Work ($\dot{W}$) has been expressed in terms of kilogram-meters per kilogram of body weight per second and air velocity as W_V^3. Data (– – –) taken from Hill (8) are shown (see text).

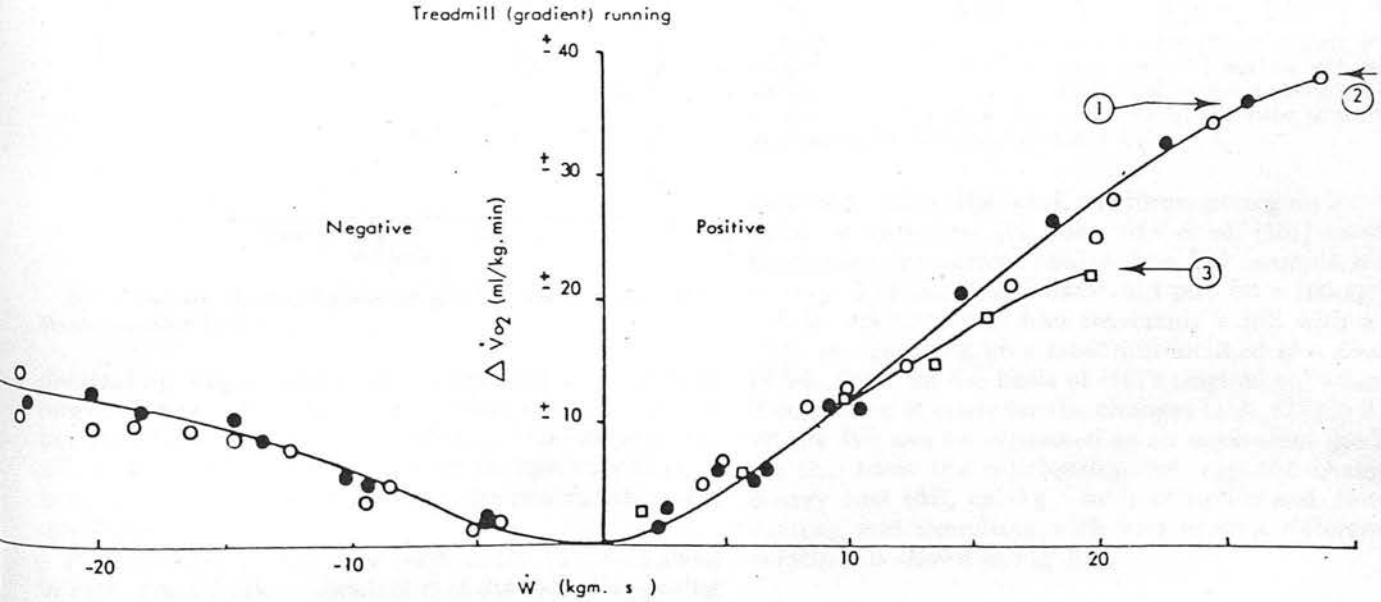

FIG. 5. Change in aerobic cost ($\Delta \dot{V}O_2$) of running uphill and downhill at different speeds on a motor-driven treadmill. Work ($\dot{W}$) with and against gravity is expressed in kilogram-meters per second. Symbols as Fig. 3.

However, beyond a W_V equivalent to ~12 m/s the apparent $\dot{W}$ diminishes and the complete relationship between $\dot{W}$ and W_V^3 over the range of W_V studied in this investigation is better described by the following quadratic equations ($P < 0.001$)

($kg \cdot m \cdot kg^{-1} \cdot s^{-1}$)

$= 0.00034\ W_V^3 = 0.029 \times 10^{-6}\ (W_V^3)^2$; $\quad r = +0.986$

Gradient running at minimal W_V. The results for gradient running at the same values of V as for the "wind" experiments, but with $W_V < 1\ m \cdot s^{-1}$ are shown in Fig. 5. The data have been plotted in terms of $\Delta \dot{V}O_2$ (calculated from a base line of running for a given V at zero gradient) against the lifting work ($\dot{W}$, $kg \cdot m \cdot s^{-1}$) performed. For all three subjects during uphill the $\Delta \dot{V}O_2/W$ relationship was linear up to levels of work

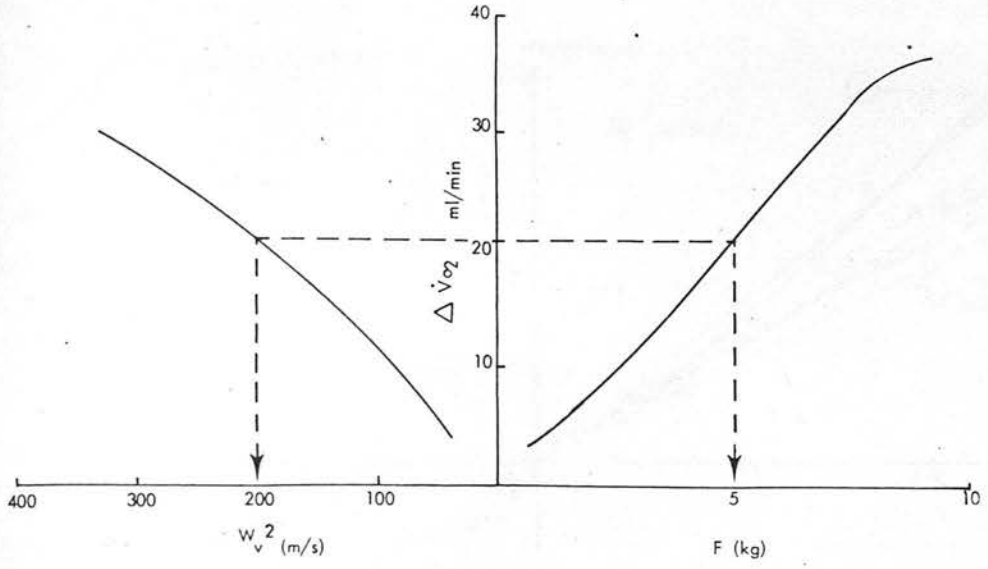

FIG. 6. Change in oxygen intake ($\Delta\dot{V}O_2$, $ml \cdot kg^{-1} \cdot min^{-1}$) against W_V^2 (see Fig. 3) and horizontal component of force (F) for work against gravity (see Fig. 5). Data shown are for *subj 2*. Values of F for each subject were estimated in this way for W_V above and below 15 $m \cdot s^{-1}$ and plotted against W_V^2. Calculated regression (α) coefficients are summarized in Table 1.

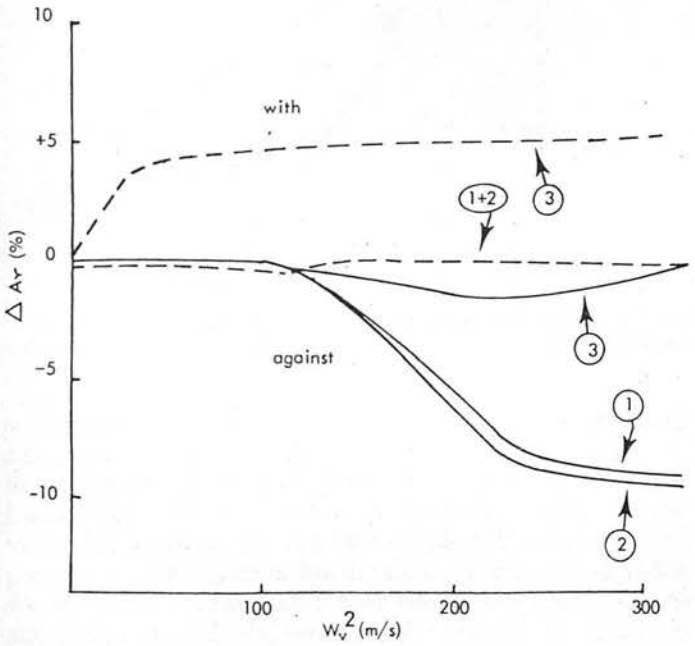

FIG. 7. Percent change of projected area (A_r) with increasing W_V^2. Mean values for 3 subjects.

demanding $\dot{V}O_{2\,max}$ and was independent of V. During downhill running the increase in $\Delta\dot{V}O_2$ was reduced and beyond a negative W of ~15 $kg \cdot m \cdot s^{-1}$ the slope of the $\Delta\dot{V}O_2/W$ line diminished markedly, though as with positive work the association between the two variables was unaffected by V.

Projected areas and drag coefficients. The data given in Figs. 3 and 5 allow calculation of the total F opposing the runner if comparison is made at the same V (Fig. 6). The resulting slope of the F/W_V^2 gives an estimate of α for human subjects and thus allows the C_D to be calculated (see METHODS, *Eq. 1*) for the three subjects at W_V above and below 15 $m \cdot s^{-1}$ (see METHODS and Table 1) provided the projected areas (A_r) are known. The changes of A_r with W_V are shown in Fig. 7 and the mean C_D data at high and low W_V are summarized in Table 2.

Apparent efficiency of work with and against wind and gravity. To calculate the apparent efficiency of work

TABLE 2. *Work efficiency of subjects*

W_V, $m \cdot s^{-1}$	Subj No.	A_r, m^2	A_r/A_D	A_r/H^2	α, $kg \cdot W_V^2$	C_D
1–15	1	0.456	0.256	0.153	0.026	0.91
	2	0.475	0.253	0.138	0.026	0.88
	3	0.430	0.242	0.136	0.022	0.82
Mean		0.454	0.259	0.142	0.024	0.87
>15	1	0.417	0.234	0.140	0.019	0.73
	2	0.432	0.230	0.126	0.015	0.56
	3	0.428	0.241	0.135	0.011	0.42
Mean		0.425	0.235	0.133	0.015	0.57

Values are given for the projected areas (A_r) in relation to body surface area (A_D) and body height squared (H^2) together with the drag coefficients (C_D) for running against wind velocities above and below 15 $m \cdot s^{-1}$. α is the regression coefficient for the slope of the F/W_V^2 relationship for 3 subjects (see METHODS).

with and against the wind, the forces acting on the body must be visualised [cf. Margaria et al. (10)] as either facilitating or retarding progression. For example, a force of (say) 5 kg exerting a backward pull on a 100-kg man will be equivalent to him traversing a hill with a 1:20 slope or exercising on a treadmill inclined at a gradient of 5%. Thus, on the basis of Hill's original equation and if correction is made for the changes in A_r (Table 2, Fig. 7), the W_V^2 can be expressed as an equivalent gradient. On this basis the relationship between the change of energy cost (ΔE, $cal \cdot kg^{-1} \cdot m^{-1}$) of uphill and downhill running and exercising with and against different air velocities is shown in Fig. 8.

DISCUSSION

The consistency of the oxygen cost of running results and their agreement with previous published work (5) are regarded as evidence of the accuracy of the $\dot{V}O_2$ measurements and the calibration and recording of airflows in the wind tunnel. The reproducibility of the data also overcomes the possible criticism that two different treadmills were used in the present investigation (Fig. 1). The projected areas (A_r) and drag coefficient (C_D) of the

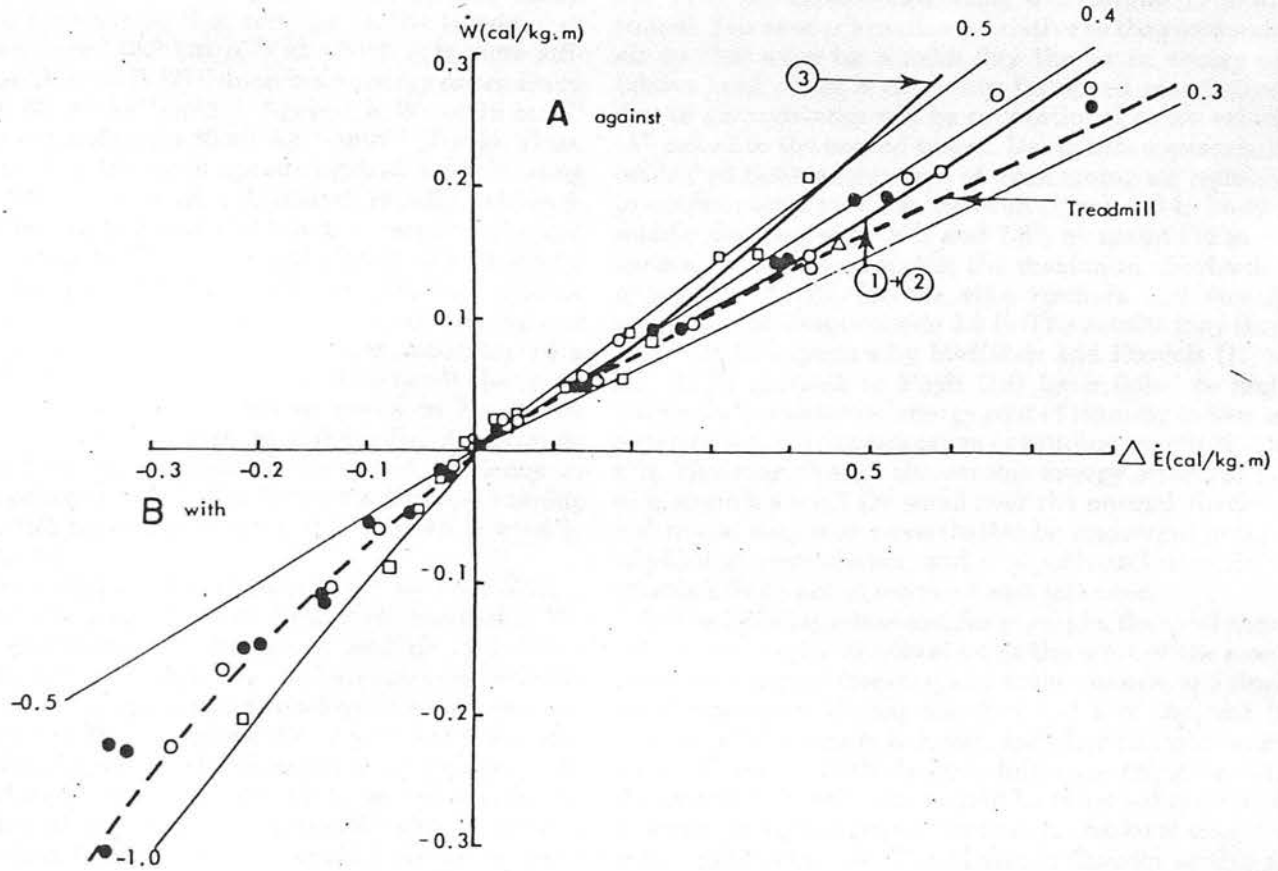

FIG. 8. Comparison between change of energy cost (E, cal·kg^{-1}·m^{-1}) between uphill and downhill treadmill work (- - -) and running against and with a wind (subject symbols as in Fig. 3). Wind velocity has been expressed as an equivalent gradient (see text).

subjects during the running experiments show close agreement with Hill's (8) original observations on an 8-in. model in the wind chamber at the National Physical Laboratory. He found that A_r in the running posture varied as 0.146 of the model's height (H) to the second power and the C_D from his data can be calculated as 0.9, which compares favorably with the mean values of 0.143 and 0.89, respectively, found in the present investigation (Table 1). Pugh (14) found A_r to be 0.266 times the body surface area (A_D); the value for this study is 0.251. The corresponding ratio for A_r to H^2 and A_D for running with the wind for which, to the author's knowledge, no previous data are available, are 0.147 and 0.259.

The energy expended to overcome air resistance at W_V <15 m·s^{-1} is closely in agreement with that given by Hill (8) and from the data given in Table 2 the following equation, which represents the horizontal force (F) acting on the body (at <15 m·s^{-1}), can be derived: $F = 0.024\ W_V^2$; or if A_r is taken into account, $F = 0.053\ W_V^2\ A_r$. The equation is approximately the same as Hill's and is consistent with some earlier observations reported by du Bois-Reymond (1). At high airflows the present data find some agreement with those of Pugh (13, 14), but there are fundamental differences between his work and this investigation. Pugh concluded from the results of this study that the change in energy cost of running against different W_V was dependent on V and that exercise on a gradient was always less efficient than the "equivalent" work against air resistance. His observations are difficult to reconcile with the data given in Figs. 2–4 and 8. Provided W_V does not exceed 15 m·s^{-1} a precise equivalence can be drawn between horizontal and vertical forces: the increased (or decreased) O_2 cost per kilogram body weight and per meter of distance covered is the same and independent of V. The subject exercised with the same "apparent" and net mechanical efficiencies in both situations. The lack of association between $\Delta\dot{V}O_2$ and V is not difficult to appreciate during treadmill work at different W_V, because in this form of exercise the effective (forward) speed of progression is zero. At minimal airflow all the energy consumed is utilized as internal work raising and lowering the center of gravity and altering the kinetic state of the limbs and little or none appears as external (useful) work. Thus an increase in treadmill speed (V) would be expected to effect an overall rise in the aerobic energy cost of running, but this should not affect that portion necessary to overcome a given W_V provided A_r is unchanged, the $\dot{V}O_{2\,max}$ of the subject is not exceeded, and the runner's style remains constant. The present results suggest that these conditions are met except at the highest wind velocities.

At $W_V > 15$ m·s^{-1}, the $\Delta\dot{V}O_2/W_V^2$ relationship appears to depart from linearity (Fig. 3) and there is an apparent decrease in C_D (Table 2). These changes are associated with a reduction in A_r (Fig. 7) but the change is small (~10%; Fig. 7). A reduction of this order of magnitude would account for ~7% change in drag (D). The estimated change in D is of the order of 60% (Table 2). The influence of $\dot{V}O_{2\,max}$ on the $\Delta\dot{V}O_2/W_V^2$ relationship is more difficult to assess. Undoubtedly it has a confounding

influence on the results of *subject 3* (see Figs. 3, 4, and 8). It must be appreciated that running on the treadmill at the slowest speed (8.3 km·h^{-1}) at which it is more efficient to run than walk (2) demands an energy expenditure of at least 35 ml·kg^{-1}·min^{-1}. Against a W_V of 18 m·s^{-1} the added aerobic cost is 30 ml·kg^{-1}·min^{-1} (Fig. 3). Thus, even at modest treadmill speeds against an increasing W_V, the $\dot{V}O_{2\,max}$ of most subjects is rapidly achieved. *Subject 3* ran at 11.3 and 12.9 km·h^{-1}, respectively, and clearly reached his $\dot{V}O_{2\,max}$ at the higher W_V. *Subject 3* also differed in two other respects from his more athletic counterparts. He was less efficient at level running and even at minimal W_V his natural style incorporated a forward flexion of the upper body. The small change of his A_r with increasing W_V can be noted in Fig. 7. His smaller range and increase in $\Delta\dot{V}O_2$ (Fig. 3) certainly contributed to his "apparent" increased efficiency of running against the wind (Fig. 8) but his different running style may (for reasons given below) have been an equally decisive factor.

Subjects 1 and *2* were characterised by high $\dot{V}O_{2\,max}$ values and the leveling off of $\Delta\dot{V}O_2$ with increasing W_V occurred (for them) at submaximal aerobic work levels (Fig. 2). However, at high W_V the two subjects radically changed their running styles and adopted a body posture more similar to that described for *subject 3* at lower W_V. They lowered their heads, changed their (upper) body angle, and leaned into the wind. Thus, as well as reducing A_r as noted above, they were probably able to convert potential drag to body lift. This would have the effect of decreasing both the positive and negative done within each stride (9) and thereby reduce the energy cost. At the highest W_V studied the increased lift was subjectively noticeable and the two subjects reported a feeling of flying between strides and being raised on their toes so that the normal heel-toe contact of running was diminished. A conversion of drag to lift would be expected to reduce aerobic cost both directly and indirectly. The direct effect would be expected to reduce C_D (Table 2) and the increased bounce of the body may contribute indirectly to the amount of work that can be performed by muscles (without recourse to aerobic metabolism) due to phenomenon of elastic recoil (3), which is known to occur at high running speeds or in conditions of excessive body lift.

Running with a W_V produced opposite results to those found against a wind but the reduction in energy cost was much less for a following wind than the increase found for a head wind (Fig. 3). For example, with a head W_V at 15 m·s^{-1}, $\Delta\dot{V}O_2$ was 24.4 ml·kg^{-1}·min^{-1} compared with −11.9 with the same following wind, a saving of approximately 50% of the expected value. Again the reasons are clear: on a motor-driven treadmill the wind will only assist the runner to a certain limited extent. As the wind velocity increases beyond the speed of the treadmill belt the runner will have to brake progressively to maintain his position on the mill and the proportion of negative work within each stride will increase. Thus, working with a following wind is precisely analogous to downhill (negative work) running on a treadmill in calm air.

In outdoor running of course conditions will be different to those experienced using a treadmill in a wind tunnel. The runner's motion is relative to the surrounding air so that even on a calm day the extra energy cost (above that which is necessary for speed maintenance) due to air resistance will be proportional to his velocity (V) raised to the second power. Using this approach, the estimated extra energy cost of overcoming air resistance in outdoor track running has been calculated to be 4% at middle distance (6 m·s^{-1}) and 7.8% at sprint (10 m·s^{-1}) speeds. At marathon speeds the maximum effect will be approximately 2% for the elite runners and virtually negligible for times outside 2.5 h. The results may therefore help to explain why McKiken and Daniels (11; see Fig. 1) in contrast to Pugh (13) have failed to find a difference between the energy cost of running indoor and outdoor over the normal range of running speeds (2–5 m·s^{-1}). However, though the aerobic energy effects of running against a wind are small over the normal physiological range, they may nevertheless be important in terms of physical performance, and it is perhaps instructive to examine them not in terms of cost but time.

In the 100-m sprint event, for example, the total energy requirement is for acceleration at the start of the race to overcome inertial forces, speed maintenance, and finally wind resistance. During the first 2–3 s of the race the velocity of the runner is lower, and thus the wind resistance will have relatively little influence compared with the last 60 m. If resistance could be removed completely, it would be equivalent to running on the level compared with a gradient of 4%. The minimum time for an elite (10 m·s^{-1}) athlete would be decreased by approximately 0.25–0.5 s. In the marathon event (best recorded time, 2 h 8 min 33.6 s), the air resistance on a calm day is equivalent to a slope of ~1%; if this was abolished the athletes' speed would be expected to increase by 0.82 km·h^{-1}. This would reduce his time by over 5 min!

The effects of a following wind for reasons outlined will have approximately half the effect, though presumably unlike the treadmill a low air speed will assist the forward velocity of the runner outdoor. However, once the following wind exceeds the velocity of the runner, he would have to perform an increased amount of negative work in the latter half of each stride to maintain his running posture. Thus, when running on an oval track on a windy day, an athlete will find it difficult to achieve his best performance. The extra energy expended against the wind (say) in the back straight will not be compensated by an equal gain in the home straight. If for example we have a wind blowing (W_V) down the track equal to the runners speed (V) in a middle-distance race (6 m·s^{-1}), in the back straight the backward pull on the athlete's body will be approximately 3.8 kg, equivalent (for a 65-kg athlete) to running up a gradient of 5.8%. This will increase his oxygen cost by a ~4 ml·kg^{-1}·min^{-1} or reduce his speed by 1 km·h^{-1}. In the home straight his forward speed will be equivalent to the W_V and this resistance to progression theoretically zero. To compensate for the extra energy expanded in the back straight he would need W_V of at least 15 m·s^{-1}. Athletes are also sensitive to cross winds and running the bends with W_V at right angles to the runner would also be expected to increase energy expenditure. Hill (8) has

suggested that where $W_V = V$ the extra cost of running the crown of the bend will (paradoxically) be 1.41 times greater than the head resistance, i.e., $\sqrt{(1 + W_V^2)/(V^2)}$. Thus the overall effect on performance would likely to be an increase in time of approximately 4 s per lap. A race on a track under such windy conditions would be similar to an undulating course experienced by cross-country and long-distance road running athletes. On a calm day, the relative air velocity will be equivalent to the runners' speed (6 $m \cdot s^{-1}$) at every point on the track if air resistance could be eliminated the performance time (using the same argument as above) would be decreased by 1.6 s per lap. Clearly the most sensible way for an athlete to run a race on an oval track on a calm or windy day is to shield behind a front runner until the closing stages of the race. We performed some crude shielding experiments in the wind tunnel and they generally confirmed the results reported by Pugh (14). The actual magnitude of effect was solely dependent on the proximity of the two runners. On the treadmill it was quite easy to reduce the effects of air resistance by at least 80–85% by shielding. If these data are applied to a track race on a calm day at a V of 6 $m \cdot s^{-1}$, then the saving in time would be approximately 1 s per lap. As pointed out by Pugh (14), this is in accord with common observations and experience of middle-distance runners.

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Metabolic Cost of Exercise and Physical Performance in Children with Some Observations on External Loading

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Summary. The metabolic cost ($\dot{V}O_2$) of running was studied on a motor-driven treadmill in nine athletic boys, five athletic girls, and nine active boys aged 11–13 years and the results compared with their performance times during racing out of doors. On 15 of the children, additional observations of the effects of external loading on aerobic power output were made. The results showed that $\dot{V}O_2$ was proportional to body weight in children but when expressed in ml · kg^{-1} · min^{-1}, $\dot{V}O_2$ for a given speed of running was significantly higher in children than expected from previously collected data on adults. There were no significant differences between aerobic cost of running of the athletic boys, girls, or the active boys. The increased $\dot{V}O_2$ ml · kg^{-1} · min^{-1} in children appeared to be independent of stride length and frequency but external loading equivalent to 5% of body weight reduced $\dot{V}O_2$ (ml · kg^{-1} · min^{-1}), particularly at the higher speeds. It was suggested in young active and athletic children due to their relatively light body weights and highly developed aerobic power outputs, that the required frequency of leg movement was not optimally matched to the force necessary to produce the most economic conversion of aerobic energy into mechanical work. Thus, in competitive events their performance times were related to their maximal aerobic power output ($r = -0.75$) but their times were always inferior to those which one might have expected from previous aerobic power weight data collected on adult male and female athletes.

Key words: Oxygen intake – Children – Running treadmill exercise – External loading – Performance

There have been several studies of the physiological responses of healthy children to exercise (Åstrand and Rodahl 1970) but data on the pre-adolescent child, particularly for those engaged in athletic activity, are sparse. Following

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our investigations of adult male and female endurance athletes (Davies and Thompson 1979) an opportunity presented itself to study a small group of pre-adolescent boys and girls who trained regularly and specialised in long distance (3,000 m and cross country) athletics. In healthy school children of this age, Åstrand (1952) has reported high values of maximal aerobic power output ($\dot{V}O_2$) when account is taken of body weight ($ml \cdot kg^{-1} \cdot min^{-1}$) but relatively low levels of running efficiency during standardised work on a motor-driven treadmill. This latter observation, which is surprising when one considers that children are able to pedal a stationary bicycle ergometer with the same efficiency as adults (Godfrey et al. 1971), has recently been confirmed by Silverman and Anderson (1972). A further interesting factor is that whereas in adult endurance athletes running performance and $\dot{V}O_{2\,max}$ appear to be closely matched and integrated (Davies and Thompson 1979), cross-sectional studies of children (Åstrand 1952; Davies et al. 1972) have shown that although $\dot{V}O_{2\,max}$ expressed in $ml \cdot kg^{-1} \cdot min^{-1}$ remains fairly constant, performance improves markedly during early adolescence. A solution to these problems may rest in the relationship between the applied force and frequency of (leg) movement of running in lightweight children with highly developed aerobic power outputs. It is well known in man (Wilkie 1950) that the force a muscle develops is inversely proportional to its speed of shortening and for the optimal conversion of energy into mechanical work these two factors must be kinetically balanced. It is conceivable that in athletic children who are capable of running at relatively high velocities this condition is not met and economy of effort is sacrificed for speed. Therefore, to investigate this problem further, the relationship between the aerobic cost of running under standardised conditions on a motor driven treadmill and performance times on an outdoor running track has been studied on 23 pre- and adolescent children, 15 of whom performed additional experiments with external loading corresponding to 5% and 10% of their body weight.

Subjects and Methods

The physical characteristics and maximal aerobic power outputs of the 23 children, sub-divided into active and athletic boys and athletic girls, are shown in Table 1. The athletic children were recruited mainly from the Dartford Athletic Club.

The aerobic cost of running on the treadmill at zero gradient and various ($8-16\,km \cdot h^{-1}$) speeds (V) was determined using an open circuit technique. Expired air was collected via a low resistance valve and smooth bore tubing into a Tissot spirometer and analysed for O_2 and CO_2 content using physical analysers. Maximal aerobic power output ($\dot{V}O_{2\,max}$) was determined during uphill treadmill running using criteria previously outlined (Davies 1968). The external loading experiments were performed on separate occasions on 15 of the children using a specially designed weight jacket. The subjects ran on the treadmill under normal conditions and then repeated the experiments with added loads equivalent to 5% and 10% of their body weights respectively. The design of the weight jacket allowed the weight (in the form of sand) to be evenly distributed in small pockets which covered the upper body. During all experiments step frequency (S_f) was measured manually using a stop-watch and counting foot contact on the treadmill belt. Stride length (S_l) was calculated from V and S_f. The performance times of the children for 400 m, 800 m, 1,500 m, and 3,000 m were obtained by questionnaire and from the official records of the schools athletic association.

Table 1. Physical characteristics and maximal power outputs. Age, weight, height, maximal ventilation ($\dot{V}_{E\,max}$), aerobic power output ($\dot{V}O_{2\,max}$), and cardiac frequency ($f_{H\,max}$). Mean (± SD) data

Subjects	Age (years)	Weight (kg)	Height (cm)	$\dot{V}_{E\,max}$ BTPS ($l \cdot min^{-1}$)	$\dot{V}O_{2\,max}$ STPD		$f_{H\,max}$ ($beats \cdot min^{-1}$)
					($l \cdot min^{-1}$)	($ml \cdot kg^{-1} \cdot min^{-1}$)	
Athletic ♂ ($n = 9$)	12.6 ± 1.1	36.43 ± 6.71	147.3 ± 9.6	92.64 ± 13.08	2.54 ± 0.43	69.9 ± 2.9	208 ± 7
Active ♂ ($n = 9$)	12.2 ± 0.4	37.84 ± 5.13	148.4 ± 10.1	87.52 ± 12.48	2.36 ± 0.32	62.2 ± 2.0	213 ± 7
Athletic ♀ ($n = 5$)	13.8 ± 0.7	38.62 ± 7.20	152.0 ± 5.7	96.02 ± 16.48	2.45 ± 0.25	64.6 ± 8.2	211 ± 5

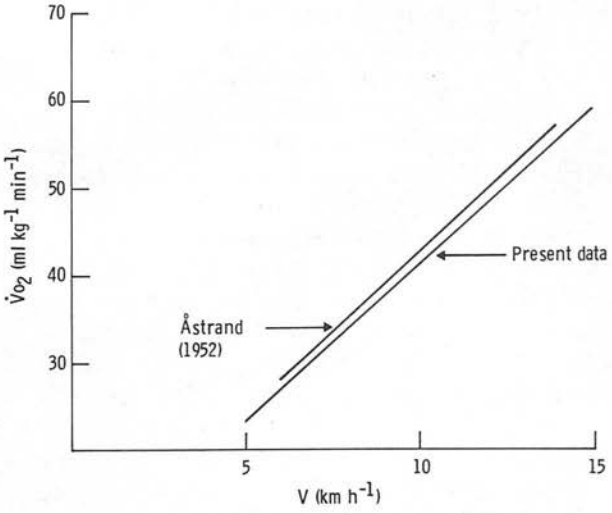

Fig. 1. Oxygen intake ($\dot{V}O_2$) in relation to speed (V) of running at zero gradient on a motor-driven treadmill

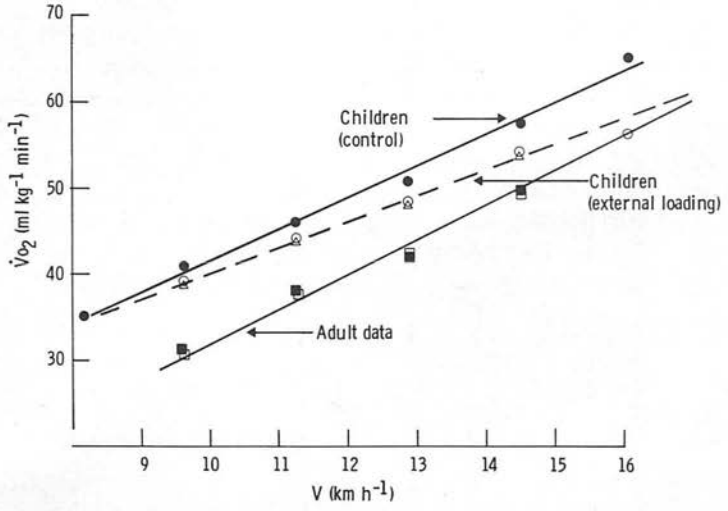

Fig. 2. The effect of external loading on the metabolic cost of running in children. Mean data ($n = 15$). (●) control; (○) external loading equivalent to 5% body weight; (△) external loading equivalent to 10% body weight. Mean data ($n = 3$) for similar observations on adult subjects are shown. (■) 5% and (□) 10% added body weight. The regression lines shown for the control data are taken from Fig. 1 (see text) for children, and Davies and Thompson (1979) for adults. The regression equation for loading ≡ 5% body weight in children is given by: $\dot{V}O_2$ ml · kg^{-1} · min^{-1} = 11.102 + 2.980 V (km · h^{-1}); $r = +0.90$. The slope and intercept of external loading regression line is significantly ($p < 0.05$) different from the childrens' and adult control data

Results

The metabolic cost ($\dot{V}O_2$) of running at different speeds (V) in the 23 children investigated is shown in Fig. 1. The $\dot{V}O_2$ for given speed was not significantly different ($p > 0.1$) in the boys and girls and the combined relationship between

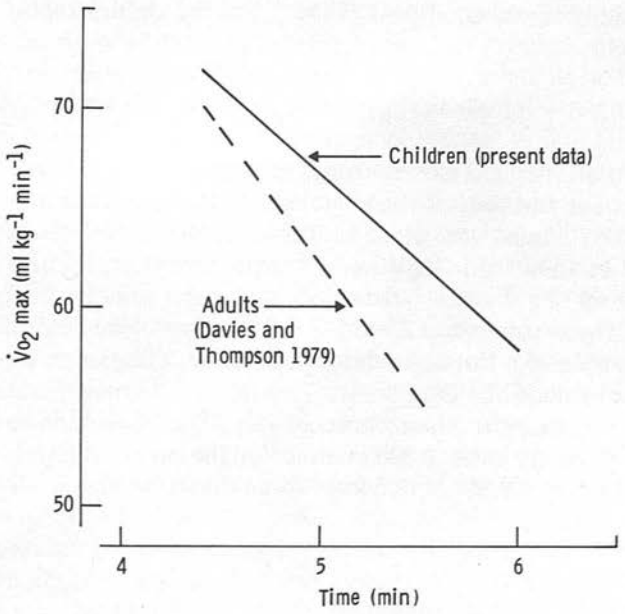

Fig. 3. The relationship of maximal aerobic power output ($\dot{V}O_{2\,max}$) to 1,500 m time recorded on the track. The regression lines shown are calculated from data of the boys in the present study and adult male athletes observed previously (Davies and Thompson 1979). A correction factor of 1.115 was used to convert the originally recorded adult athletes 5 km speed to enable comparison to be made with the children at the shorter (1.5 km) distance. The conversion is in accord with the relationship between the current world record speeds and unpublished observations by the author, for the two distances. The regression equation for the ♂ children is given by $\dot{V}O_{2\,max}$ (ml · kg^{-1} · min^{-1}) = 111.33 − 8.894 (1.5 km time-min), $r = -0.748$ and for the ♂ adults $\dot{V}O_{2\,max}$ (ml · kg^{-1} · min^{-1}) = 129.87 − 13.480 (1.5 km time-min); $r = -0.854$

the two variables could be represented by the following linear regression equation:

$$\dot{V}O_2\ (\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = 5.714 + 3.541\ V\ (\text{km} \cdot \text{h}^{-1}),$$
$$r = +0.95; \quad n = 122\,.$$

Using individual regression equations and expressing the $\dot{V}O_2$ (ml · kg^{-1} · min^{-1}) in terms of a set speed of 15 km · h^{-1} ($\dot{V}O_{2_{15}}$) for each child it was found that the aerobic cost of running under these conditions was independent of stride length (S_l; $r = -0.39$; $p > 0.5$) and stride frequency (S_f; $r = +0.27$; $p > 0.5$). The application of external loading to 15 of the 23 children studied did not significantly change their style of running in terms of S_l and S_f, but it did have a marked effect on $\dot{V}O_2$. External loading equivalent to 5% of body weight significantly ($p < 0.05$) decreased the slope and increased the intercept of the $\dot{V}O_2$ relationship. However, the addition of further weights ($\equiv 10\%$ body weight) did not have any further effect on the relationship (Fig. 2).

The absolute maximal aerobic power outputs ($\dot{V}O_{2\,max}$) of the children were proportional to body weight:

$$\dot{V}O_{2\,max}\ (l \cdot min^{-1}) = 0.258 + 0.0589\ wt\ (kg), \quad r = +0.90; \quad p < 0.001\ .$$

The positive intercept corresponded closely to the expected resting oxygen intake for normal healthy boys and girls of the same age. The mean values of $\dot{V}O_{2\,max}$ expressed in $ml \cdot kg^{-1} \cdot min^{-1}$ are given in Table 1. The athletic boys had an average value of 69.9 $ml \cdot kg^{-1} \cdot min^{-1}$ compared with 64.6 $ml \cdot kg^{-1} \cdot min^{-1}$ and 62.2 $ml \cdot kg^{-1} \cdot min^{-1}$ recorded in the girl athletes and active boys, respectively. These values are 25–35% higher than those found previously on a random sample of a London school population, (Davies et al. 1972). The highest recorded values of $\dot{V}O_{2\,max}$ were 73.7 $ml \cdot kg^{-1} \cdot min^{-1}$ and 73.5 $ml \cdot kg^{-1} \cdot min^{-1}$ in a boy and girl athlete, respectively. The best guide to $\dot{V}O_{2\,max}$ in the boys was given by their 1,500 m time on the track: $\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) = 111.33 − 8.894 (1.5 km time-min); $r = -0.748$ (Fig. 3).

Discussion

The maximal aerobic power outputs of the athletic boys and girls in this study are comparable with those found in adult marathon and ultra-long distance runners (Davies and Thompson 1979). The values for active children are greater than those given in the literature for American (Adams et al. 1961), European (Pavizkova 1968), and British children (Davies et al. 1972). However, they do compare with data observed by Åstrand (1952) for more active Swedish children. The highest individual values of $\dot{V}O_{2\,max}$ for the boys and girls recorded in this investigation were 73.7 $ml \cdot kg^{-1} \cdot min^{-1}$, respectively; the latter figure should be compared with the highest figure (77 $ml \cdot kg^{-1} \cdot min^{-1}$) so far recorded on an adult female athlete (Bergh, pers. comun.).

During submaximal work, the combined regression equation for $\dot{V}O_2$ against speed for the three groups studied, is the same as that given by Åstrand (1952). Thus, children do have a higher aerobic cost for a given distance covered when standardised for body weight than adults (Fig. 2; Davies and Thompson 1979). From the results of his study, Åstrand suggested that the "apparent" low running efficiency may be a function of their body size. Children are naturally smaller than adults and have shorter legs which will necessitate, for a given speed of running, a higher working rate in terms of stride frequency. This is certainly true, and when observing children running one is immediately aware of their higher "striking" rate, but nevertheless, if Åstrand is correct, one might expect some direct relationship between $\dot{V}O_2$ and stride length or frequency in children and adults, and this clearly is not the case. In this study $\dot{V}O_2$ at a given speed in children was not significantly related to either of these variables. The weight jacket experiments would suggest that it is not body size but body mass which is important and this factor interacts with velocity of leg movement to produce a given efficiency in children.

Hill (1939) showed from his work on frog muscle, that for the optimum conversion of chemical energy to mechanical work the force applied and speed of movement must be matched. In his experiments he found that for the highest efficiency to be attained, ideally, the force must be one half and the speed one quarter of their respective maximal values. Wilkie (1950) later confirmed and applied Hill's findings ((1939) to human (forearm) muscle and it is now generally accepted that for most ergometer work optimal efficiency is acquired in normal subjects at submaximal loading using one movement/s (60 rpm). However, in treadmill running the velocity of movement is fixed by stride length and the speed of the belt and the forces applied are governed by body weight. Theoretically one would only expect uneconomic movement in running to arise when a subjects' potential for (muscle contraction) speed is not matched by his body mass. This appears to be borne out in the present study; external loading reduced the $\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$) and increased the "apparent" efficiency of running in young athletic children particularly at high imposed speeds (Fig. 2). At the highest speed the children could sustain with 5% or 10% of added body weight the O_2 cost of exercise approached that of the adult athletes previously measured in my laboratory (Davies and Thompson 1979). This suggests that the muscle of the young are not intrinsically less efficient than those of adults but during running, particularly at middle distance speeds, light-weight children cannot effectively match their load to the imposed speed of (leg) movement. The fact that when children pedal a bicycle ergometer, and body weight is therefore carried, they do so with the same mechanical efficiency as adults, (Godfrey et al. 1971) strongly supports this argument. However, some caution is necessary.

It will be noted that the addition of weights had little effect at low speeds and increasing the external load from 5%–10% of body weight had no additional effect on $\dot{V}O_2$ (expressed in $ml \cdot kg^{-1} \cdot min^{-1}$). We have not previously observed that light-weight men are generally more or less efficient at running than their heavier counterparts or indeed that light-weight small adult female athletes run less economically than men (Davies and Thompson 1979). Adding (Fig. 2) or reducing (pers. observ.) weight is without effect on O_2 cost ($ml \cdot kg^{-1} \cdot min^{-1}$) of exercise in men. Clearly, the effects of adding weights are subtle and it may be that only in children with a high maximal aerobic power output to weight ratio and the capacity to sustain relatively high speed of running are the effects of external loading clearly observed. It should also be noted that the observations on external loading are of no practical value with regard to improving performance of young athletic children. The addition of weights in young children may improve their economy of effort from a physiological viewpoint but it does not enable the child to run faster. However, the results may help to explain the relatively inferior performances of children when compared with young adults and why performance shows such a marked improvement during adolescence.

Fig. 3 shows that, as in adult athletes, performance time in children on the track is closely related to $\dot{V}O_{2\,max}$. The regression coefficient is the same order of magnitude for the two groups but association between the two variables is displaced to the right in children. For a given $\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) a child

athlete will produce a slower time for a fixed distance than his adult counterpart. Maintaining the same pace in a child demands a higher aerobic energy expenditure than an adult. As the child grows and moves through adolescence his performance will improve (provided his training is maintained) as a result of an increase in the economy of movement. The expected improvement in the aerobic cost of running due to kinetic balancing will be of the order of $8\ ml \cdot kg^{-1} \cdot min^{-1}$ (Fig. 2), which is equivalent to $1.5\ km \cdot h^{-1}$. In the present study the best boy and girl were able to run the 1,500 m in 4.63 min and 4.96 min respectively, which on the above basis would mean an improvement of 20 s and 35 s on their times. This would still leave the boy and girl some 300–400 m behind the current world record holders for the 1,500 m event. To reach the world record times would require a 15% improvement in the $\dot{V}O_{2\,max}$ (Table 1 and Fig. 3) which even if possible, would require intensive training for a long period of time. It is not surprising that athletes seldom reach their peak before early adulthood.

Finally, it is perhaps noteworthy that although the top boy and girl athletes have the same $\dot{V}O_{2\,max}$ their performance times differed by about 7%. This is less ($\sim 3\%$) than the sex related difference in performance observed in adult elite athletes, but it does suggest that the difference in potential physical (as opposed to physiological) performance in "endurance" runners may manifest itself earlier in childhood than one had supposed, possibly before the onset of adolescence. This is clearly an area where more data are required and research indicated.

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Effect of Air Resistance on the Metabolic Cost and Performance of Cycling

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Summary. The metabolic cost ($\dot{V}O_2$) of cycling against a range of wind velocities (V_W) was studied in a wind tunnel of 15 male cyclists and the results compared with work on a stationary cycle ergometer, uphill cycling on a treadmill, and performance times in road racing competitions.

The results showed that $\dot{V}O_2$ at constant treadmill speed was proportional to V_W^2 and was a linear function of work rate, both on the stationary ergometer and during uphill cycling on a motor driven treadmill. Maximal values of 5.04 $l \times min^{-1}$ (69.3 $ml \times kg^{-1} \times min^{-1}$) and 482 W were observed. The mean force (F) acting on the cyclists during the experiments in the wind tunnel was found to be equal to 0.0175 $V_W^2 \times ms^{-1}$ ($r = +0.98$). The mean projected area (A_P), drag areas (A_D) and drag coefficient (C_D) for the 15 cyclists were 0.50 m^2, 0.280 m^2, and 0.56 m^2, respectively. During cycling out of doors on a calm day $\dot{V}O_2$ was calculated to be a curvilinear function of the cyclists' speed of progression (V). The best guide to the cyclists' maximal aerobic power output ($\dot{V}O_{2\,max}$) was given by their 16.1 km (10 mile) time:

$$\dot{V}O_{2\,max}\ (l \times min^{-1}) = -4.219 + 0.7727\ V\ (ms^{-1})\ r = +0.89\ .$$

The results suggested that the relative aerobic power output (% $\dot{V}O_{2\,max}$) which could be sustained for a given time by elite cyclists, is similar to that found previously for marathon athletes. However, due to the differences in the non-drag component of the work for given metabolic cost the cyclist will travel approx. $2^1/_2$ times the distance of an endurance athlete.

Key words: Cycling – Wind resistance – Aerobic performance – Treadmill exercise – Projected area

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It is well known that the aerobic power output during exercise at a constant speed against air resistance is proportional to the square of the wind velocity (V_W^2 – Pugh 1970), though it has been shown that for practical purposes when running out of doors under normal conditions the effect of air resistance is small and may be neglected (McKiken and Daniels 1976). However, in cycling where frictional (rolling) resistance is small, the work against air resistance is considerable and theoretically could account for almost all of the metabolic cost of the exercise. It is therefore surprising, that the physiological responses to cycling against given wind velocities under standardised conditions have not been systematically studied. Nonweiler (1956) has investigated the forces acting on a cyclist mounted on a bicycle suspended in a wind tunnel, and Whitt (1971) and di Prampero et al. (1979) have developed equations of motion for a cyclist based on their own (in the latter case) and earlier estimates of overall tractive resistance (Chandler and Chandler 1910) from towing experiments. In addition, there have been fragmentary reports of the energy cost of subjects pedalling touring bicycles at relatively low rates (for a general review, see Adams 1967) but only Pugh (1974) to the author's knowledge, has contributed data for racing cyclists over a wide range of speeds. Unfortunately, Pugh's observations were made on the runway of a disused airfield which was not level, and this fact may have confounded his results. In order to overcome such difficulties the present study was conducted on racing cyclists who rode their bicycles on a motor driven treadmill which was housed inside a wind tunnel. The cyclists performed work at a set treadmill speed against various wind velocities and the results compared with their times in various road racing competitions.

Subjects and Methods

The physical characteristics of the racing cyclists are given in Table 1. They were all members of the Farnborough and Camberley Cycling Club. They were all amateurs and none was of international standard. The experiments were carried out at the Institute of Aviation Medicine, Farnborough.

The cyclists were required to pedal their bicycles at a set speed of 4.7 ms^{-1} on a motor driven treadmill against wind velocities varying from 1.5–18.5 ms^{-1}. The treadmill was housed in a wind tunnel and an air stream produced by a fan was deflected through an angle of 90° by a set of vertical vanes 3 m in front of the treadmill. The air flow was reasonably laminar at low velocities but was turbulent at the higher fan settings: vane anemometer readings varied by at least 1 ms^{-1} across the tunnel. The subjects cycled continuously on the level for 8 min against a given air speed and oxygen intake ($\dot{V}O_2$) was measured during the final 3 min by the standard (Douglas bag) open circuit technique. In five subjects the effects of several different air speeds were studied but in the remaining ten subjects observations were limited to two settings of the fan. In separate experiments 12 of the subjects performed treadmill work on the level and up various gradients (range 2°–8°) at a constant speed (4.7 ms^{-1}) and with minimal (< 0.25 ms^{-1}) wind velocity. Five of these subjects also performed similar work on a stationary cycle ergometer.

For all experiments the protocol and work time were similar. The maximal aerobic power output ($\dot{V}O_{2\,max}$) for each cyclist was determined using criteria previously described (Davies 1968). The frontal projected area (A_P) of the cyclists was estimated from photographs taken during the experiments.

Calculations from the raw data were made in two ways. The mean drag force (D) acting on the cyclist as a result of wind velocity (V_W) is:

$$D = kV_W^2 . \quad (1)$$

Table 1. Physical characteristics of the 15 cyclists and a comparison of the maximal aerobic power output ($\dot{V}O_{2\,max}$) of five of the subjects who performed maximal work on a stationary cycle ergometer in addition to cycling uphill on a motor driven treadmill in the presence of minimal ($< 0.25\ ms^{-1}$) wind speed

Cyclists	Age (year)	Height (cm)	Weight (kg)	Uphill treadmill		Stationary cycle	
				$\dot{V}O_{2\,max}$		$\dot{V}O_{2\,max}$	
				($l \times min^{-1}$)	($ml \times kg^{-1} \times min^{-1}$)	($l \times min^{-1}$)	($ml \times kg^{-1} \times min^{-1}$)
$n = 5$	24.4 ± 13.2	175.3 ± 10.1	69.5 ± 7.2	4.66 ± 0.48	67.1 ± 4.4	4.62 ± 0.47	66.5 ± 3.9
$n = 15$	25.4 ± 12.1	177.4 ± 7.6	69.0 ± 5.9	4.40 ± 0.47	63.7 ± 6.3	–	–

When the cyclist is moving at a constant ground speed (V), the mechanical power ($\dot{W}$) necessary to overcome the drag is:

$$\dot{W} = DV = kV_W^2 V . \quad (2)$$

In this study with a treadmill housed in a wind tunnel, V was constant and equal to 4.7 ms^{-1}, thus $W = k_1 V_W^2$ where $k_1 = k \times 4.7$. Thus, when cycling on the treadmill at a constant speed, the extra oxygen intake ($\Delta \dot{V}O_2$) due to the wind is given by:

$$\Delta \dot{V}O_2 = \frac{\dot{W}}{e} = \frac{k_1}{e} V_W^2 , \quad (3)$$

where e is the mechanical efficiency of the exercise. The overall oxygen intake ($\dot{V}O_2$) is given by:

$$\dot{V}O_2 = \dot{V}O_2^0 + \frac{k_1}{e} V_W^2 , \quad (4)$$

where $\dot{V}O_2^0$ is the oxygen intake for treadmill cycling at 4.7 ms^{-1} in the absence of wind. By plotting $\dot{V}O_2$ against V_W^2 and assuming an efficiency of 0.25, k_1 and thus k (which equals $k_1/4.7$ and represents the absolute value of the coefficient relating drag to wind velocity) was calculated and from Eq. (1) the mean drag force (D) acting on the cyclist was determined.

The second method used for calculating k was based on the same principles as described above but was obtained by constructing graphs relating $\dot{V}O_2$ to V_W^2 and to the mechanical power output ($\dot{W}$) of the cyclists recorded during stationary ergometer work. Values of V_W^2 and $\dot{W}$ at equal $\dot{V}O_2$ were determined, $\dot{W}$ was divided by the velocity (V) to obtain the total force (F) opposing motion and k was calculated as the slope of the F/V_W^2 regression line. The drag coefficient (C_D) and the drag area (A_D), which is the value of A_P when $C_D = 1$, were calculated from the formula: $C_D = D/0.5 \times d\, V_W^2 \times A_P$ where d is the air density and A_P the frontal projected area of the cyclist.

The second method has the additional advantage of showing the existence of an intercept on the F axis which at $V_W = 0$ may be interpreted as the rolling resistance (R) of cycling under the given experimental conditions:

$$F = R + kV_W^2 . \quad (5)$$

R being independent of speed. Thus, when considering cycling out of doors on a calm day ($V_W = V$), the overall external mechanical power output in level cycling ($\dot{W}$) is given by:

$$\dot{W} = FV = RV + kV^3 . \quad (6)$$

The corresponding energy expenditure can be calculated from the known relationship between $\dot{W}$ and $\dot{V}O_2$ (Fig. 1). This allows the $\dot{V}O_2$ of cycling out of doors at different ground velocities to be calculated and the cyclists' performance times (in terms of average speed) for different events to be converted to aerobic cost and related to their maximal aerobic power output ($\dot{V}O_{2\,max}$) in a way similar to that described for marathon athletes (Davies and Thompson 1979).

Results

The maximal aerobic power output ($\dot{V}O_{2\,max}$) values of the cyclists studied are given in Table 1.

The regression equation relating $\dot{V}O_2$ to V_W^2 was given by:

$$\dot{V}O_2\ (l \times min^{-1}) = 0.784 + 0.009\ V_W^2\ (ms^{-1}); r = +0.98 . \quad (7)$$

Thus, from Eq. (4), $k/e = 0.009\ ls^2 \times min^{-1} \times m^{-2} = 0.15\ mls \times m^{-2}$ and therefore $k = 0.15 \times 0.25/4.7 = 0.00798\ ml \times s^2/m^2$ or since 1 ml O_2 = 20.94 J

(assuming an $RQ = 0.96$) $k = 0.166\ \mathrm{Ns^2 \times m^{-2}}$ ($0.017\ \mathrm{kg \times s^2 \times m^{-2}}$). This may be regarded as the absolute value of the coefficient relating drag to wind velocity.

The relationship of $\dot{V}O_2$ to $\dot{W}$ during uphill cycling at a constant speed and pedalling a stationary ergometer is shown in Fig. 1.

From the relationships of $\dot{V}O_2$ to $\dot{W}$ and $\dot{V}O_2$ to V_W^2 (see Methods) the association of force (F) with V_W^2 was derived and is shown in Fig. 2. The relationship of F to V_W^2 was found to be linear over the range of V_W studied and could be represented by the following regression equation;

$$F\ (\mathrm{kg}) = 0.078 + 0.0175\ V_W^2\ (\mathrm{ms^{-1}});\ r = +0.98\ . \tag{8}$$

The close correspondence of the value for the slope of the F/V_W^2 regression line which may be taken to represent the proportionality constant k, and the derivation of the same constant from $\dot{V}O_2$ measurements above should be noted together with the value for the intercept of 78 g. This may be interpreted as representing the rolling resistance for the smooth linoleum belt of the treadmill used in the present investigation (Eq. 5).

The mean frontal (including the bicyle) projected area (A_P) for the 15 cyclists was 0.50 m^2 which gives an average drag coefficient (C_D) of 0.56 and a drag area

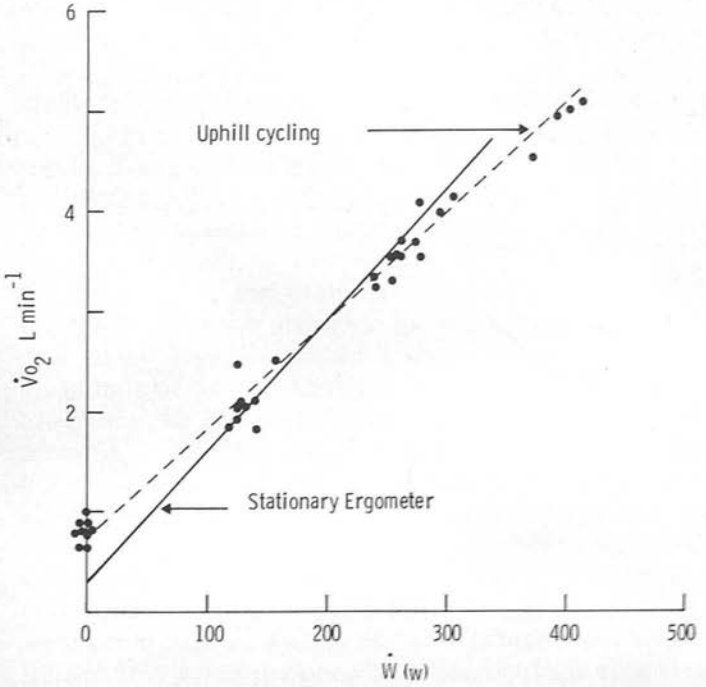

Fig. 1. The relationship between oxygen intake $\dot{V}O_2$ and work output ($\dot{W}$) during uphill treadmill cycling (●) and pedalling a stationary cycle ergometer. The respective regression equations are: Treadmill $\dot{V}O_2$ ($\mathrm{l \times min^{-1}}$) = 0.78 + 0.0106 $\dot{W}$ (W); r = +0.990. Ergometer $\dot{V}O_2$ ($\mathrm{l \times min^{-1}}$) = 0.28 + 0.0127 $\dot{W}$ (W); r = +0.993

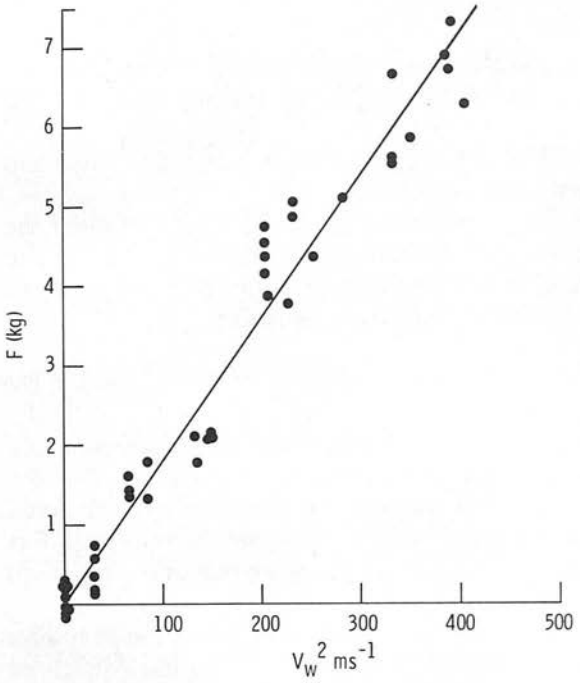

Fig. 2. The relationship between the estimated force (F) acting on the subject (see text) and the square of the wind velocity (V_W^2)

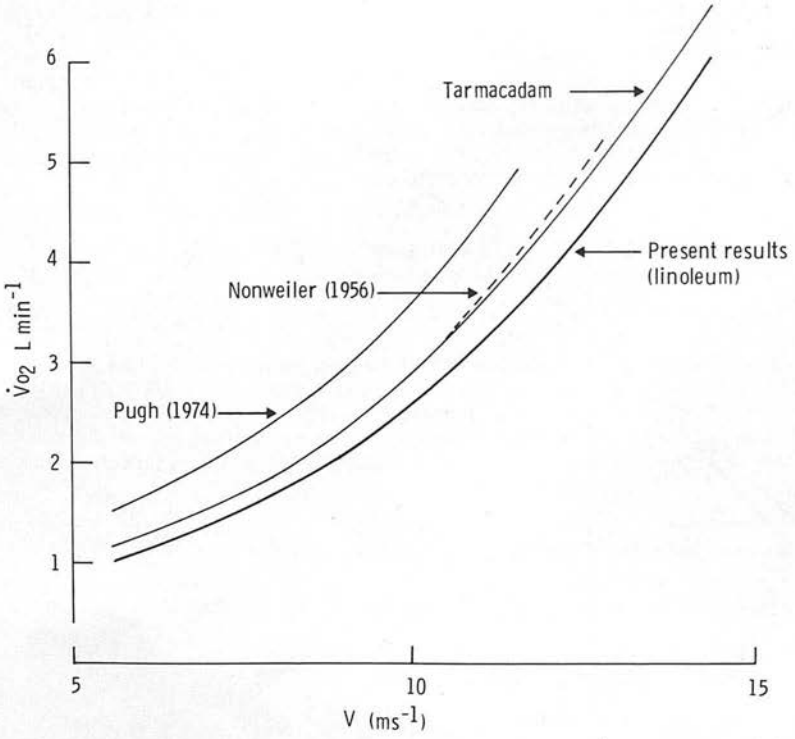

Fig. 3. The relationship between the estimated aerobic cost ($\dot{V}O_2$) and speed (V) of cycling. The present data collected on a treadmill with a linoleum belt, the estimated cost for a Tarmacadam (road) surface, together with previous calculations of Nonweiler (1956) and observations of Pugh (1974) are shown

(A_D) of 0.280 m^2. The mean ratio of A_P to body surface area (A_S) and body height to the second power (H^2) were 0.267 and 0.155, respectively.

During level cycling out of doors on a calm day the total mechanical work output is equal to the force exerted by the rider in overcoming air and rolling resistances multiplied by the ground velocity of the rider (Eq. 6). From this relationship and the data given in Fig. 1 the estimated aerobic cost of cycling for different speeds can be calculated (Fig. 3).

This allows the individual cyclist's performance times (in terms of average speed) for different events to be converted to aerobic cost and directly compared with their maximal aerobic power output measured in the laboratory. The best guide to $\dot{V}O_{2\,max}$ was given by the 16.1 km (10 mile) speed:

$$\dot{V}O_{2\,max}\,(l \times min^{-1}) = -4.219 + 0.7727\ V\,(ms^{-1});\ r = +0.89\,. \qquad (9)$$

Discussion

The results of this investigation were obtained at a constant speed of 4.7 ms^{-1} (16.9 kmh^{-1}). This was the maximum available belt speed of the treadmill housed in the wind tunnel. For normal subjects cycling out of doors, this is close to the preferred speed (Adams 1967) but for trained cyclists it was probably too slow for them to pedal at their optimum efficiency in the absence of either wind resistance or an uphill gradient. Level cycling at this speed demanded an oxygen intake ($\dot{V}O_2$) of 0.78 $l \times min^{-1}$ which represented approx. 20% of the cyclists' $\dot{V}O_{2\,max}$. One would have preferred a higher range of treadmill speeds for studying work in the absence of an imposed load but for the major part of this investigation, which involved cycling against V_W in the range of 5–18 ms^{-1} on the level and up gradients of 2°–8°, the treadmill belt speed was more than adequate to load the oxygen transport system and to cover the full range of the cyclists aerobic capacity. This factor should be borne in mind when interpreting the present results.

The major finding of this investigation is shown in Fig. 2 which summarises the calculated force acting on the subject when cycling against various wind resistances. The relationship is similar to that found by Pugh (1974) but the magnitude of the coefficients k and C_D for the cyclists in the present study is lower than he observed. These coefficients together with the calculated drag areas (A_D) are more closely in agreement with the values given by Nonweiler (1956), Whitt (1971), and di Prampero et al. (1979). For instance, Nonweiler (1956) found a mean A_D in the racing position of 0.31 m^2 and di Prampero et al. (1979) calculated k to be 0.19 $Ns^2 \times m^{-2}$. The respective mean values for this investigation are 0.28 m^2 and 0.166 $Ns^2 \times m^{-2}$. The methods used for the derivation of k, C_D, and A_D in the present study of course depends on the assumption that the cyclists are able to pedal with the same efficiency on the treadmill as when pedalling a stationary cycle ergometer. This would seem reasonable in the light of the data presented in Fig. 1. However, when considering outdoor cycling a further factor, the rolling resistance of the road surface, must also be taken into account.

From the formula of Whitt (1971) derived for racing wheels and tyres of the same dimensions as used in this study, one can calculate that the rolling resistance of Tarmacadam may be three times that for polished linoleum. Di Prampero et al. (1979) from direct towing experiments on the Monza road racing circuit found rolling resistance to be of the order of 325 g and independent of speed. This figure has been used in the calculation of the O_2 cost ($\dot{V}O_2$) of outdoor cycling given in Fig. 3. The close similarity of the oxygen intake speed relationship with the theoretical calculations of Nonweiler (1956) and Whitt (1971) is apparent as is the lack of agreement of the absolute values of $\dot{V}O_2$ measured during cycling by Pugh (1974) and earlier workers (Dill 1954; Edholm et al. 1955; Adams 1967). However, as pointed out earlier, Pugh's results are difficult to interpret and the data of the other workers cited, were collected on heavy tourist bicycles which are clearly not applicable to skilled cyclists on lightweight racing machines. If one accepts the present relationship between $\dot{V}O_2$ and speed of progression, this allows performance times to be converted into aerobic cost and to be related to maximal aerobic power output ($\dot{V}O_{2\,max}$). Unlike running (Davies and Thompson 1979), the ability to cycle at racing speeds is less closely related to $\dot{V}O_{2\,max}$ if expressed in terms of a power to body weight ratio (i.e., $ml \times kg^{-1} \times min^{-1}$), $r = +0.68$. The best guide to physical performance at cycling is given by the absolute $\dot{V}O_{2\,max}$ ($r = +0.89$). These data support the view of di Prampero et al. (1979) that cyclists with the same $\dot{V}O_{2\,max}$ ($ml \times kg^{-1} \times min^{-1}$) will perform better the greater the body mass.

The O_2 cost of cycling at a given speed is approx. (for a 65 kg man) 40% that of running, which means conversely that for equal energy expenditures you would expect a cyclist to travel $2^1/_2$ times the distance of a runner in the same time. The difference between the two forms of exercise lies mainly in the "non-drag" component of the work. In cycling and running, if account is taken of the projected (frontal) area, the work against the wind component is similar (cf. Pugh 1970). Cycling has the advantage that not only is body weight carried by the bicycle but since the legs rotate the kinetic energy of the limbs remains fairly constant. In running, the legs must be alternatively accelerated and decelerated, each cycle increasing the level of energy expenditure required. However, it is an encouraging finding that if relative ($\%\dot{V}O_2$) rather than absolute aerobic power output is considered in relation to time, rather than distance, the two forms of exercise appear to be physiologically compatible (Fig. 4) on the basis of $\dot{V}O_2$ values calculated from speed. The club cyclists of this study were able to maintain 88% $\dot{V}O_{2\,max}$ for 1 h and 61% for 4 h of cycling. These figures were less than those which could be derived for elite cyclists using their 16.1 Km (10 mile) times in conjunction with Eq. (9) to predict their $\dot{V}O_{2\,max}$. The elite cyclists data were closely similar to the results obtained on marathon and ultra-long distance (running) athletes (Davies and Thompson 1979). Finally, when considering world records it is interesting to note the difference in the computed oxygen cost of cycling on a polished (linoleum) surface and Tarmacadam shown in Fig. 3 and the possible mechanical work output which can be generated and sustained for 1 h by these elite cyclists. At world record levels the difference in $\dot{V}O_2$ is $0.35\,l \times min^{-1}$ which is equivalent to a speed of 0.28 ms^{-1}. This is close to the difference which can be found for cycling indoors (polished wood track) and out

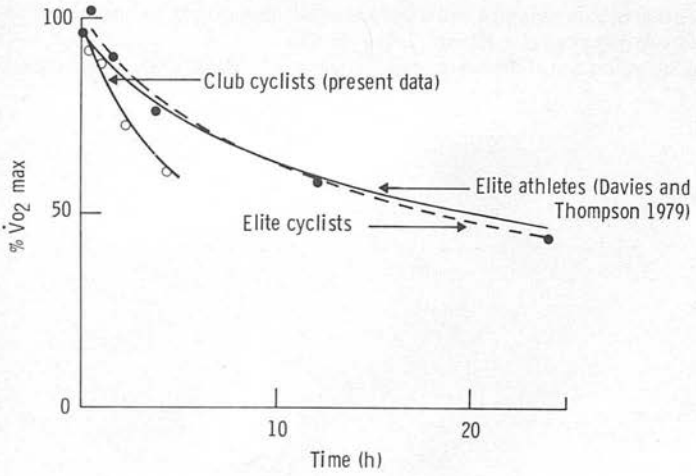

Fig. 4. The estimated relative ($\%\dot{V}O_{2\,max}$) work load the club cyclist could sustain in relation to time. For comparison previous data collected on ultra-long distance runners (Davies and Thompson 1979) and calculations for elite (world record) cyclists (see text) are shown

of doors at world record speeds. The calculated power output (assuming an efficiency of 0.25) is of the order of 500 *W*.

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